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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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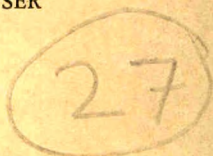
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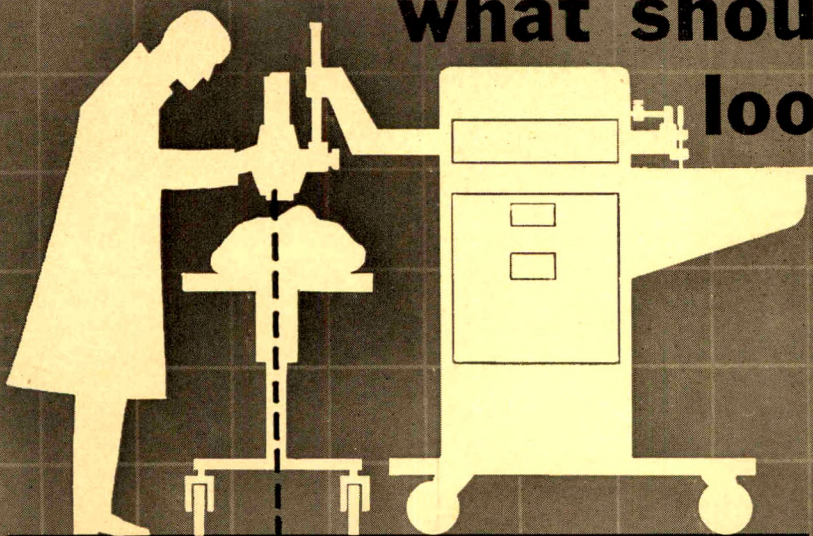


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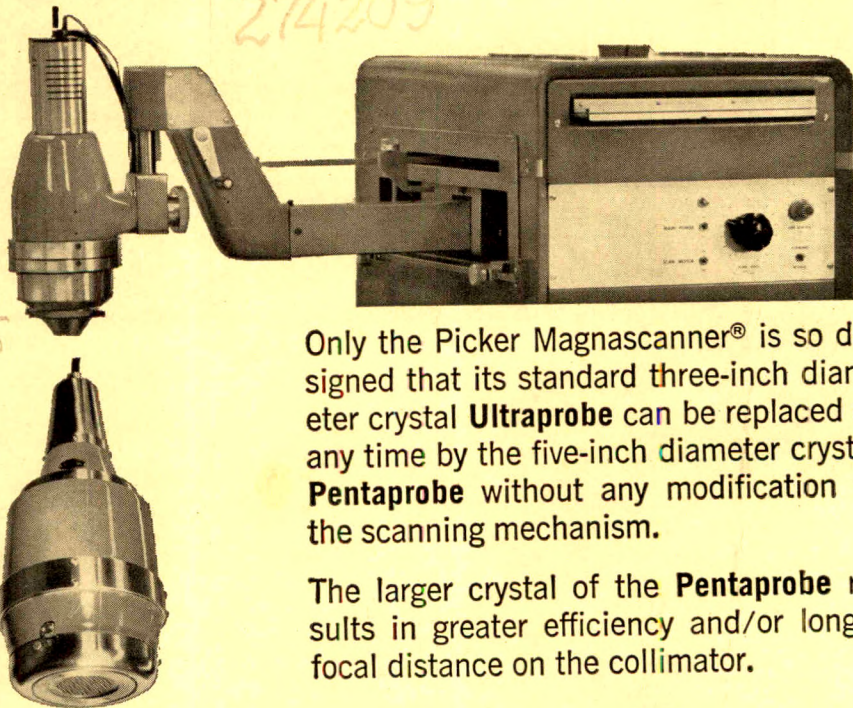
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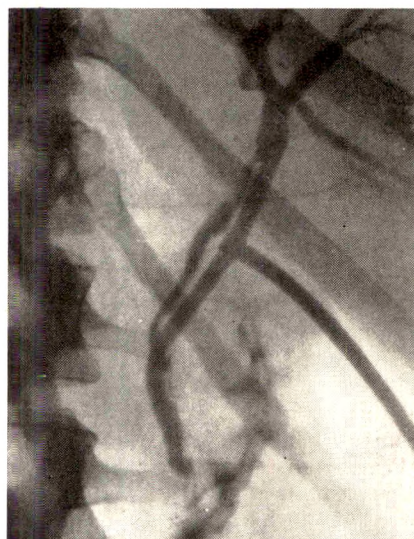
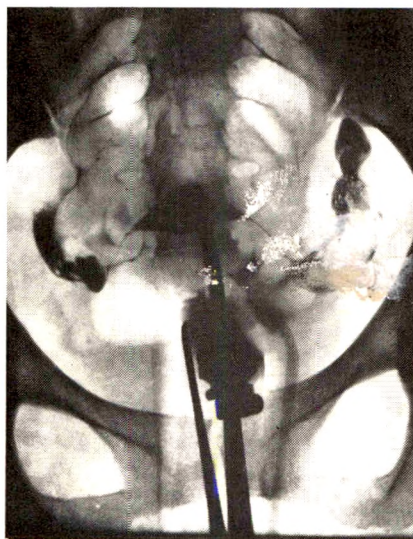
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## TABLE OF CONTENTS

<i>Skull Changes Associated with Intracranial Arteriovenous Malformations.</i> CALVIN L. RUMBAUGH, M.D., AND D. GORDON POTTS, M.D. . . .	525
<i>Dynamic Displacements of Intracranial Structures Simulating Mass Lesions.</i> MANNIE M. SCHECHTER, M.D., LAWRENCE H. ZINGESSER, M.D., AND C. CUEVAS, M.D. . . .	535
<i>The Differentiation of Extrinsic from Intrinsic Intracranial Tumors with Particular Reference to Posterior Fossa Tumors.</i> H. F. W. PRIBRAM, M.B. . . .	542
<i>Cranial and Intracranial Lesions in Neurofibromatosis.</i> KENDRICK C. DAVIDSON, M.D. . . .	550
<i>Neurofibromatosis.</i> WILLIAM T. MESZAROS, M.D., FRANCIS GUZZO, M.D., AND HILDEGARDE SCHORSCH, M.D. . . .	557
<i>Cerebral Leptomeningeal Cysts of Developmental Origin.</i> CHARLES B. WILSON, M.D., AND VURAL BERTAN, M.D. . . .	570
<i>Radiologic Detection of Astrocytoma Involving the Corpus Callosum.</i> JOHN HOWIESON, M.D., AND J. W. D. BULL, M.D. . . .	575
<i>Angiographic Findings in Agenesis of the Corpus Callosum.</i> JOHN LUDV. LARSEN, M.D. . . .	579
<i>The Roentgenology of the Meningo-Hypophyseal Trunk.</i> H. F. W. PRIBRAM, M.B., T. R. BOULTER, M.D., AND WM. F. MCCORMICK, M.D. . . .	583
<i>Tentorial Branch of the Internal Carotid Artery (Arteria Tentorii); Report of Three Cases.</i> HAJIME HANDA, M.D., JYOJI HANDA, M.D., AND MASA- HIRO TAZUMI, M.D. . . .	595
<i>Ascending Pharyngeal-Vertebral Anastomosis.</i> DEAN A. NIERLING, M.D., PAUL B. WOLLSCHLAEGER, M.D., AND GERTRAUD WOLLSCHLAEGER, M.D. . . .	599
<i>The Clinical Significance of the Azygos Anterior Cerebral Artery (A.C.A.).</i> MARJORIE LEMAY, M.D., AND CHARLES A. GOODING, M.D. . . .	602
<i>Primary Arachnoid Cysts of the Sella Turcica.</i> B. ALBERT RING, M.D., AND MARGARET WADDINGTON, M.D. . . .	611
<i>Extrasellar Extension of Pituitary Adenomas; Clinical and Neuroradiological Considerations.</i> ERICH G. KRUEGER, M.D., AND SOL M. UNGER, M.D. . .	616

(Continued on page iv)



2



<i>Arteriovenous Shunting in Cerebrovascular Occlusive Disease.</i> ERNEST J. FERRIS, M.D., JEROME H. SHAPIRO, M.D., AND FREDERICK A. SIMEONE, M.D. . . . .	631
<i>Cranial Collateral Pathways in Stroke Syndrome.</i> LEON LOVE, M.D., B. J. HILL, M.D., SANFORD J. LARSON, M.D., A. J. RAIMONDI, M.D., AND ALFRED J. LESCHER, M.D. . . . .	637
<i>Aneurysm As a Cause of Stroke.</i> WILLIAM HANAFEE, M.D., AND PETER J. JANNETTA, M.D. . . . .	647
<i>Heart Failure in Infancy Due to Arteriovenous Malformation of the Vein of Galen; Report of a Case.</i> J. STAUFFER LEHMAN, JR., M.D., KUO-YORK CHYNN, M.D., JACK W. C. HAGSTROM, M.D., AND ISRAEL STEINBERG, M.D. . . . .	653
<i>Angiography of the Posterior Cervicocranial Circulation.</i> JORGE WEIBEL, M.D., AND WILLIAM S. FIELDS, M.D. . . . .	660
<i>Post-Angiography Isotope Brain Scanning; Positive or Negative?</i> E. R. HEINZ, M.D., J. R. BRYLSKI, M.D., J. L. IZENSTARK, M.D., AND H. S. WEENS, M.D. . . . .	672
<i>Brain Abscess in Children; Radiologic Methods for Early Recognition.</i> M. TEFFT, M.D., D. D. MATSON, M.D., AND E. B. D. NEUHAUSER, M.D. . . . .	675
<i>Roentgenographic Manifestations of Complications Secondary to Cerebral Ventricular-Cardiac Atrial Shunts.</i> WILLIAM N. COHEN, M.D. . . . .	689
<i>Roentgenologic Findings in Syringomyelia and Hydromyelia.</i> DONALD L. MCRAE, M.D., AND JAMES STANDEN, M.D. . . . .	695
<i>An Anatomic, Myelographic and Cinemyelographic Study of the Dentate Ligaments.</i> BERNARD S. EPSTEIN, M.D. . . . .	704
<i>Traumatic Extradural Cysts of the Spine.</i> A. H. SHAHINFAR, M.D., AND M. M. SCHECHTER, M.D. . . . .	713
<i>Venous Intravasation of Iodized Oil during Myelography; Report of a Case with Roentgenologic Evidence of Contrast Medium in the Iliac Veins and in the Lungs.</i> RENATO RUBERTI, M.D., FRANCESCO GALLIGIONI, M.D., GIORGIO IRACI, M.D., AND ALESSANDRO NORI, M.D. . . . .	720
<i>Angiographic Findings in a Patient with Tuberous Sclerosis.</i> M. VIAMONTE, JR., M.D., R. RAVEL, M.D., V. POLITANO, M.D., AND B. BRIDGES, M.D. . . . .	723
<i>Pulmonary Manifestations of Tuberous Sclerosis.</i> ROBERT D. MILLEDGE, M.D., BARRY E. GERALD, M.D., AND WILLIAM J. CARTER, M.D. . . . .	734
<i>Roentgenographic Findings in the Renal Lesion of Tuberous Sclerosis.</i> ALEXANDER D. CROSETT, JR., M.D. . . . .	739
<i>Increased Thickness of the Fibula in Duchenne Muscular Dystrophy.</i> VIVIAN J. HARRIS, M.D., AND WILLARD S. HARRIS, M.D. . . . .	744
<i>Upright Planigrams in Pneumoencephalography.</i> GOMER W. ROBERTS, M.D. . . . .	749
<i>Officers</i> . . . . .	753
<i>Editorials</i>	
<i>The Radiologist's Responsibility for Neuroradiology.</i> COLIN B. HOLMAN, M.D. . . . .	754
<i>George W. Grier, M.D., 1882-1966.</i> LEWIS E. ETTER, M.D. . . . .	757
<i>News Items.</i> . . . .	760
<i>Book Review.</i> . . . .	763
<i>Books Received.</i> . . . .	763
<i>Statement of Ownership</i> . . . . .	764
<i>Abstracts of Radiological Literature</i> . . . . .	765

# same day re-examination in oral cholecystography obviously practical, but... diagnostically valuable?

UNSATISFACTORY opacification after routine cholecystography doesn't necessarily mean the gallbladder is diseased. Poor absorption of the contrast agent from the gastrointestinal tract, or the inability of the liver to conjugate and excrete it, might just as well be the cause.<sup>1</sup>

## 'Nonopacification' is not a condition you can treat

To determine the cause of nonopacification, most physicians request re-examination. There are two ways this has been done by the oral route in the past:

- by starting all over again at a later date — and sometimes doubling the dose of contrast agent on the second try;
- by administering more contrast agent the evening of the first unsuccessful examination (and, again, sometimes doubling the dose) and repeating the study the following day.

Both these methods share two inherent disadvantages. They take a good deal of time, and they require the patient to take a good deal of oral contrast agent. The amount given assumes clinical importance because it has been reported higher doses may result in considerable patient discomfort because of diarrhea and other significant side effects.<sup>1</sup>

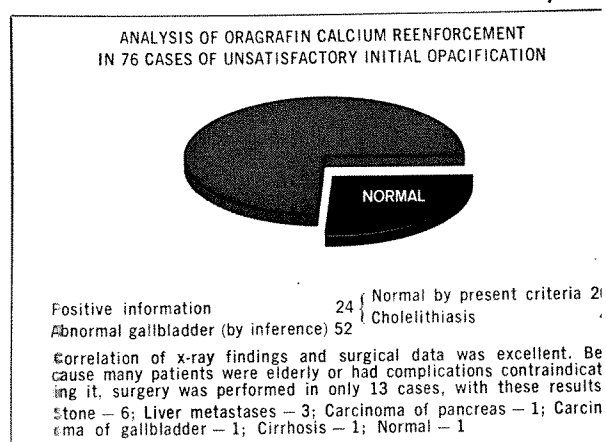
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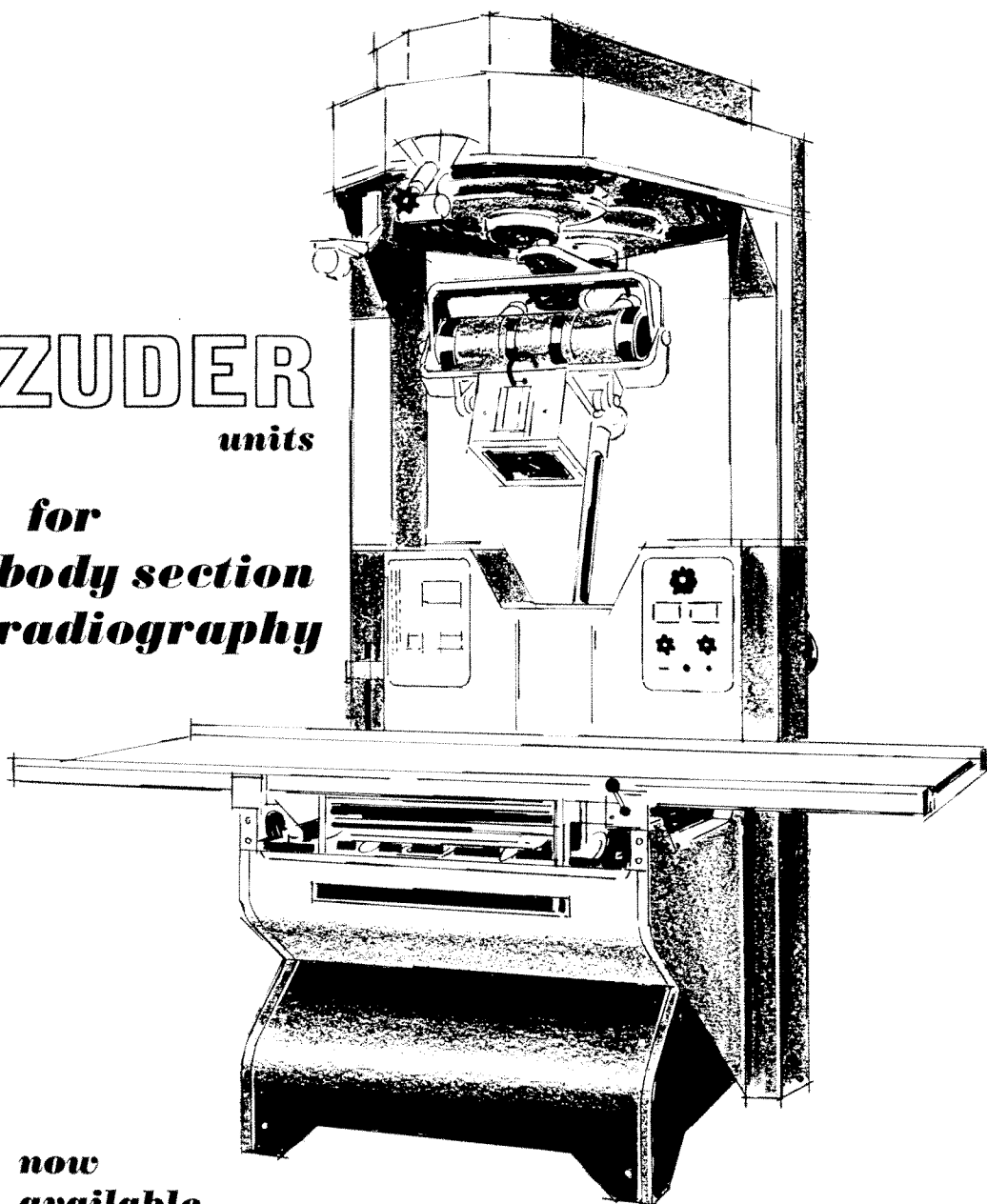
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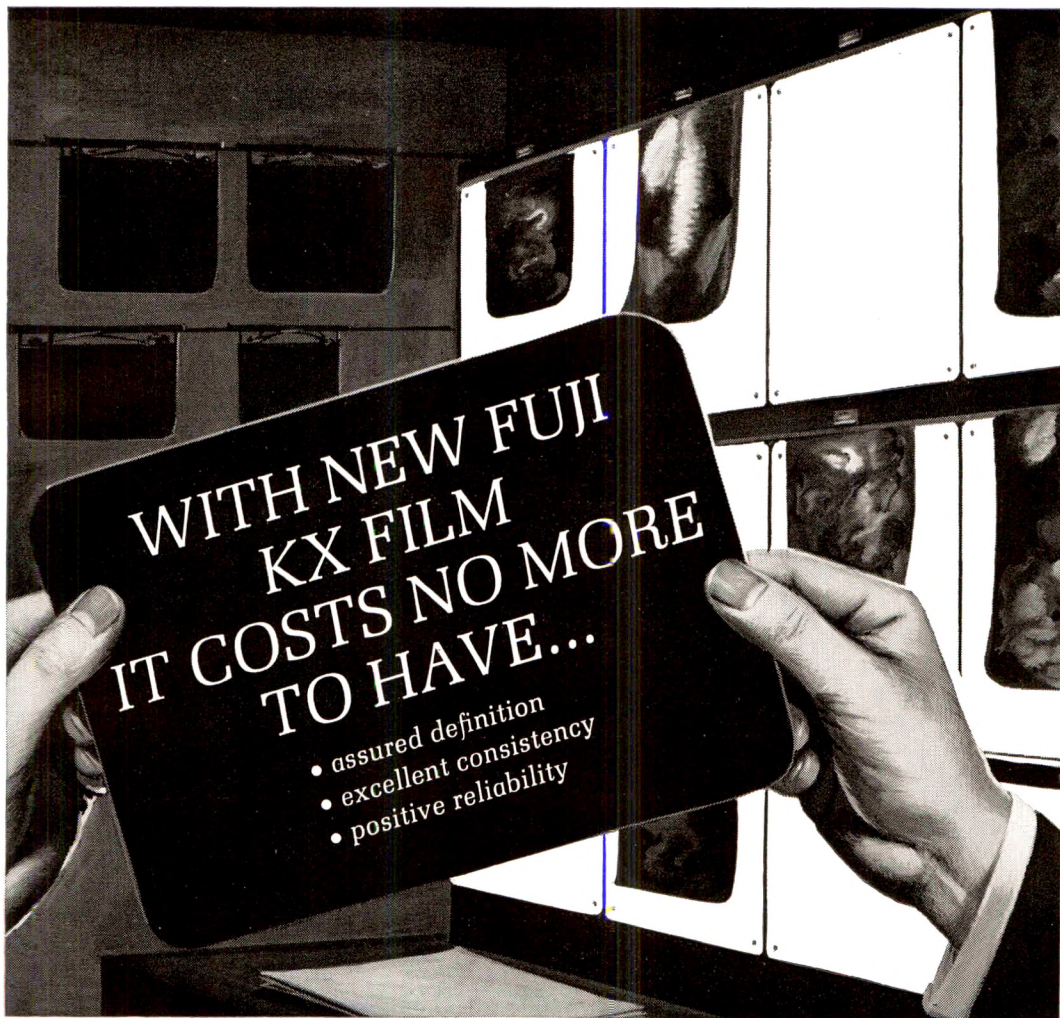


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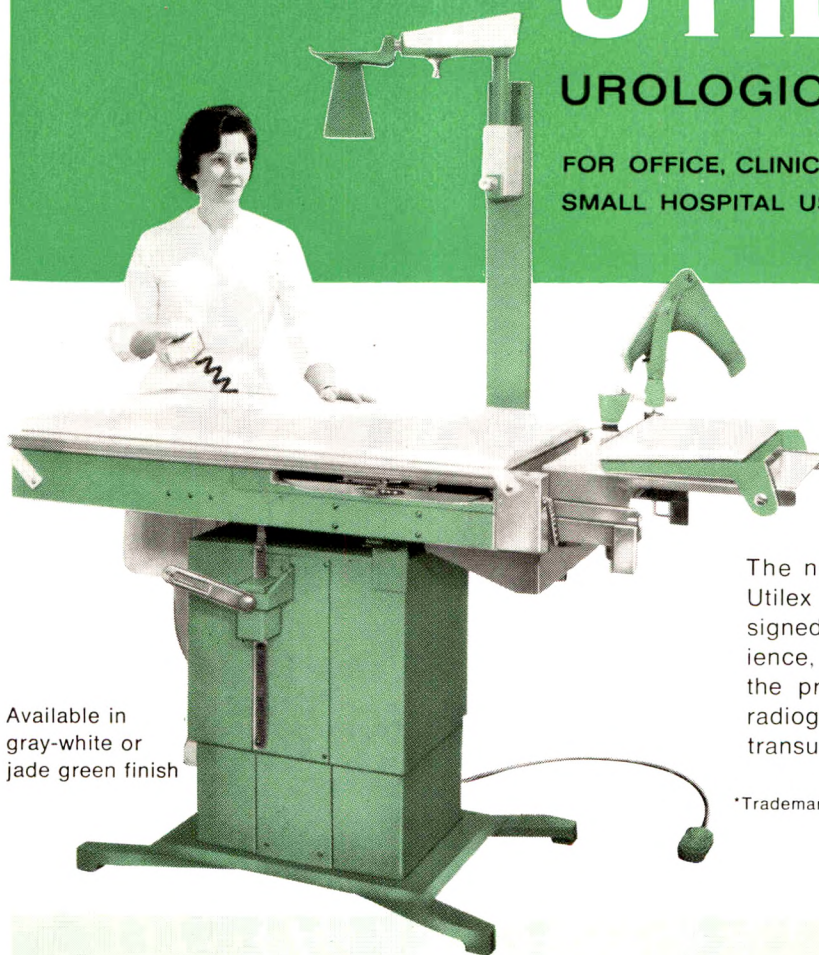


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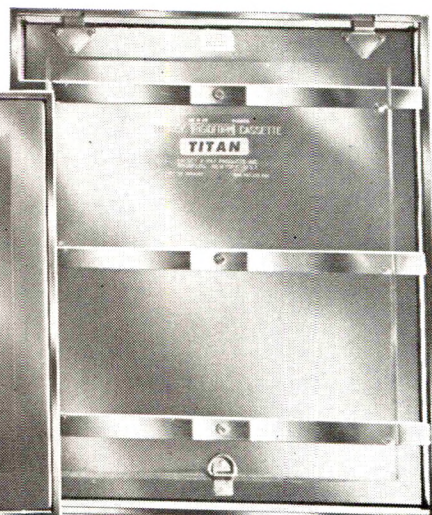
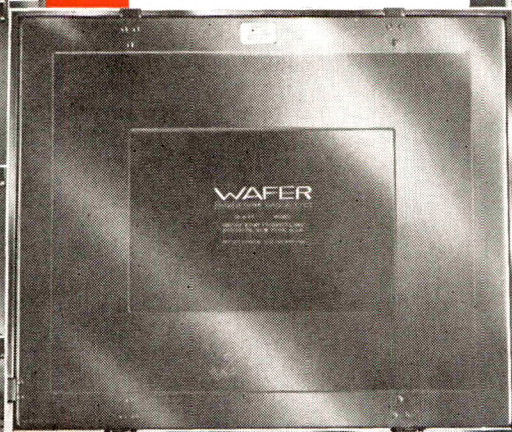
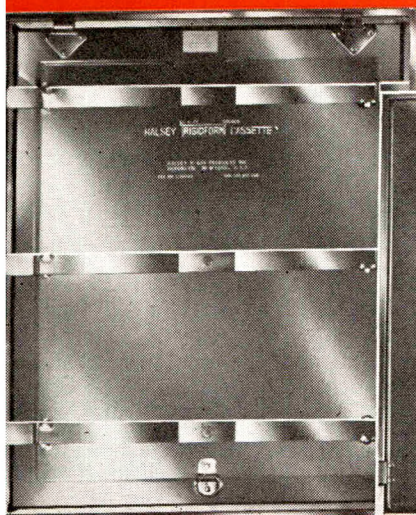
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500 ml. dilution-unit bottle holding 250 ml. Cystokon ready for dilution to desired concentrations from 30% to 15%


100 ml. multiple-dose vial to provide further concentration and volume flexibility

**Description:** Cystokon is a sterile aqueous solution containing 30% w/v of the sodium salt of 3-acetamido-2,4,6-triiodobenzoic acid. Each ml. contains 0.3 Gm. sodium acetrizoate (0.2 Gm. iodine), not more than 0.05 mg. calcium disodium ethylenediaminetetraacetate as a stabilizer, and not more than 0.15 mg. sodium phosphate as a buffer. The pH of the solution is adjusted to within 7.0 to 7.4. Since the solution itself will not support bacterial growth, additional bacteriostatic agents have not been added. The solution is somewhat sensitive to light and must be protected from strong daylight or direct exposure to the sun.

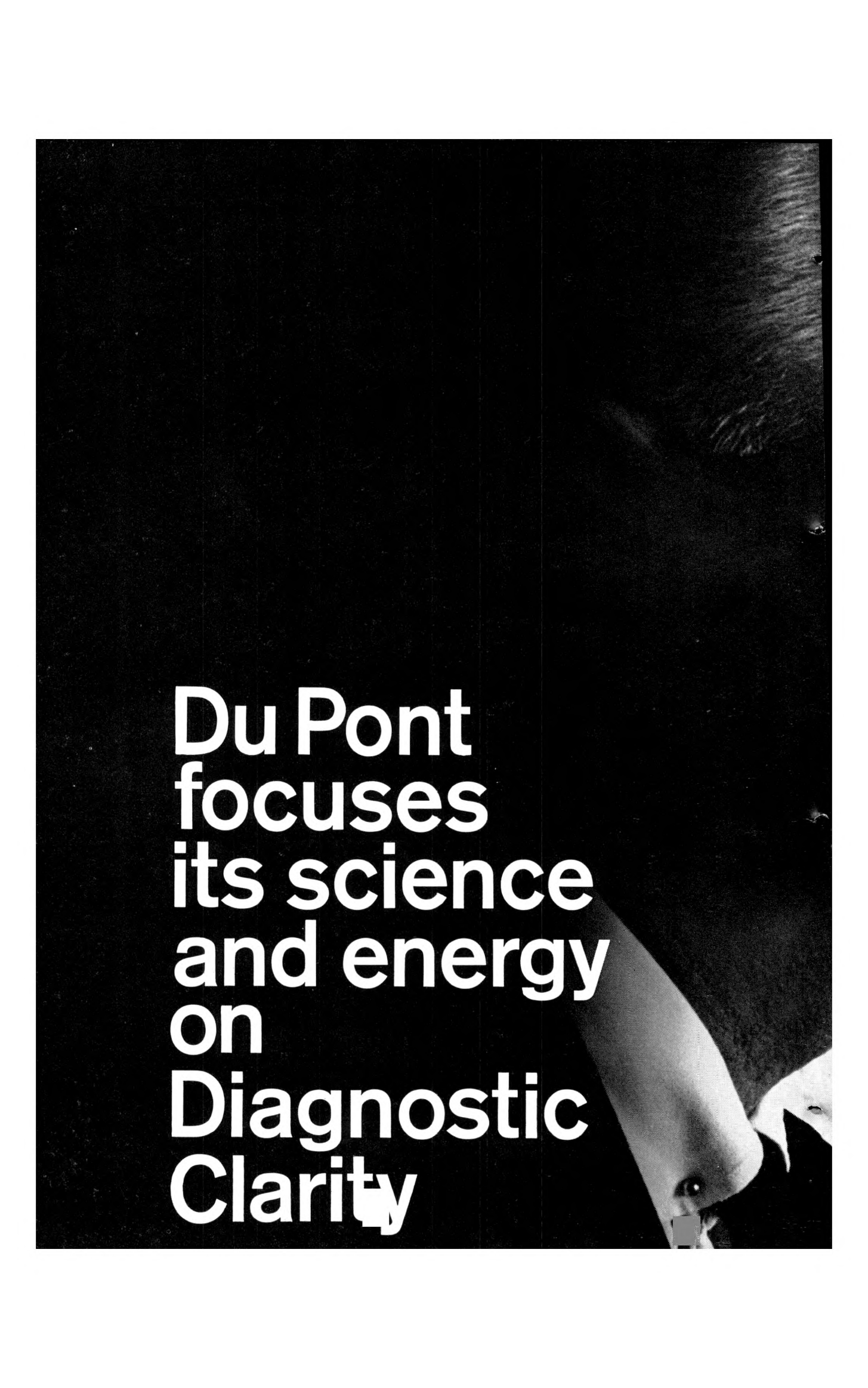
**Clinical Application:** Cystokon is prepared and packaged specifically for cystography and retrograde pyelography. It may be employed in simple retrograde cystography and pyelography, voiding cystourethrography, delayed cystography and other appropriate modifications of these techniques.

**Contraindications and Side Effects:** No evidence of irritation or toxicity to the urinary tract mucosa has been reported with Cystokon. However, minor side reactions associated with the procedure, e.g., nausea, vomiting, pain, salivation and sweating, may be encountered in rare instances. Certain pathologic conditions may, in the judgment of the attending physician, contraindicate retrograde pyelography and cystography.

**Mallinckrodt**  
Pharmaceuticals  
LINKING CHEMISTRY TO MEDICINE®

  
MALLINCKRODT CHEMICAL WORKS





**Du Pont  
focuses  
its science  
and energy  
on  
Diagnostic  
Clarity**





## Living Proof: your Technical Representative

Like the men who support him in research and manufacture, your Du Pont TR is dedicated to Diagnostic Clarity.

He is trained to respond almost instinctively to the needs of radiologists and their technologists, particularly in helping them see what they want to see.

That response takes many forms:

- ... instructing residents in fundamentals of intensifying screen technology, for example.
- ... bringing innovations firsthand to student technologists, which happened most recently with the Du Pont Bit System of technic conversion.
- ... actually rolling up his sleeves and working with your staff, as in the case of the TRs who have helped convert automatic processors to the Double Capacity system.

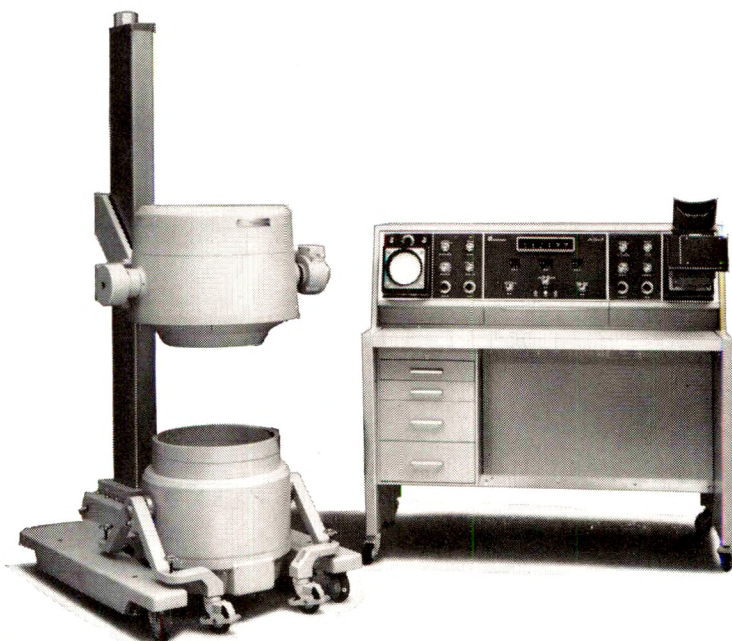
In these, and many less conspicuous ways, your Du Pont Technical Representative is living proof of our focus on Diagnostic Clarity.



**CRONEX® X-ray Products**



# Someday soon rectilinear scanners will be obsolete.



This is the new Pho/Gamma<sup>®</sup> III Scintillation  
Camera equipped for positron tomography.

## Someday is a little closer.

Pho/Gamma—clinically proved for rapid, sensitive isotope visualization in vivo. For both static and dynamic studies. And now—the new Pho/Gamma III.

Even more data from Pho/Gamma III, even more easily. Simultaneous recording and monitoring with two oscilloscopes. Visual adjustment of isotope peaks. Electronic splitting of detector output for kidney-function studies. Front-panel programming for fast-sequence cameras. Convenient desk-console housing all electronics, including the two scopes, and dual scaler-timer. Fail-safe, motorized, omnidirectional positioning of detector head. Built-in provision for system-expanding accessories, such as positron head for tomographic studies, fast digital printer, and 1600-channel multidimensional analyzer.

May we supply you with evidence of Pho/Gamma's past clinical performances—plus a look at the future of Pho/Gamma III? Consult your local Nuclear-Chicago sales engineer. Or write directly to us.

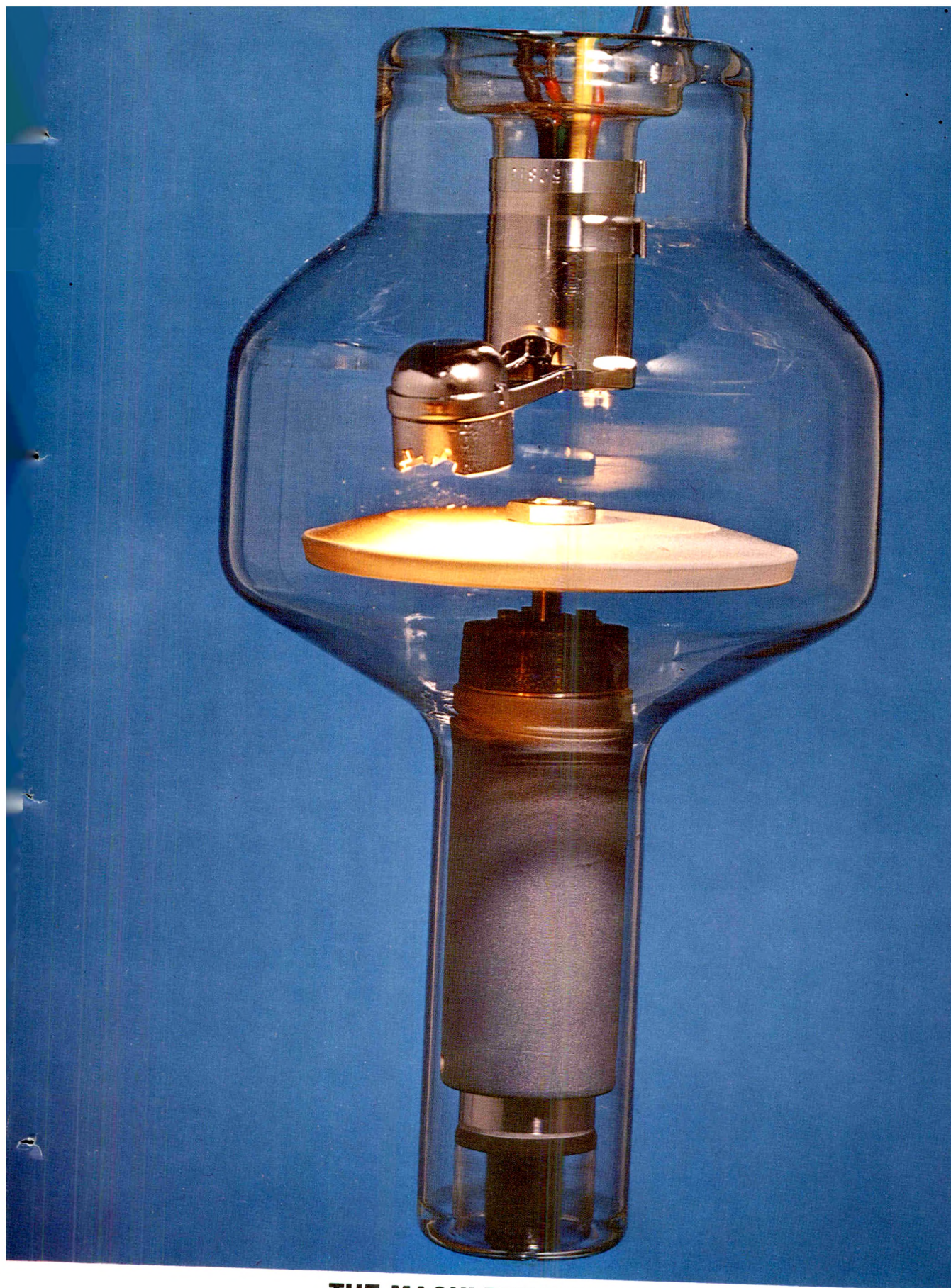
NUC D-8-224



**NUCLEAR-CHICAGO  
CORPORATION**

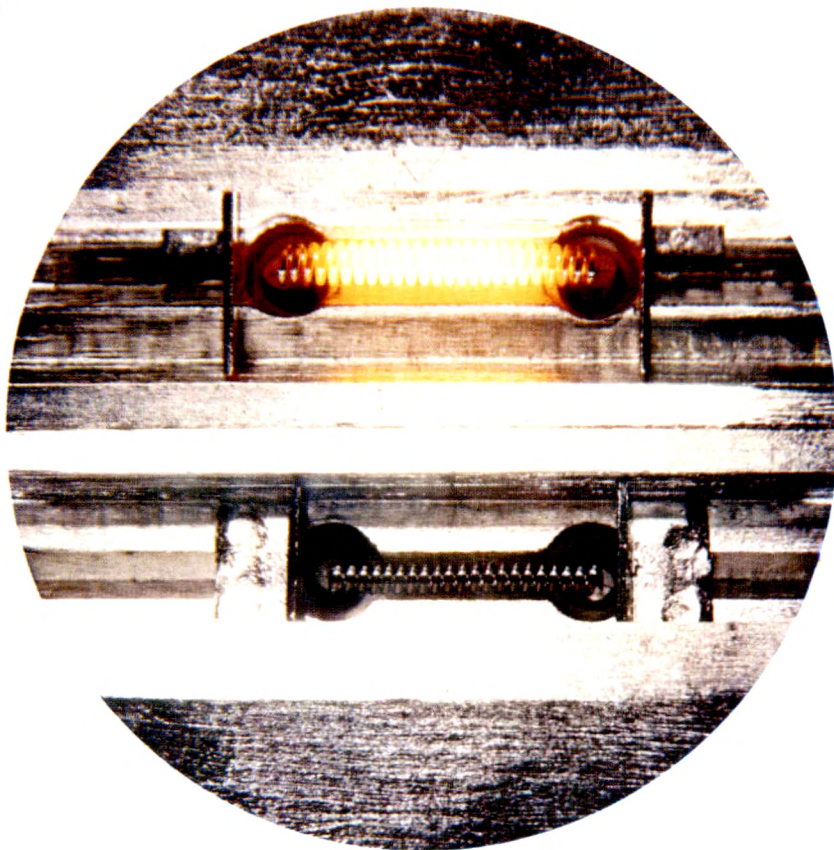
307 East Howard Avenue, Des Plaines, Illinois 60018, U.S.A. / Donker Curtiusstraat 7, Amsterdam W, The Netherlands.





**THE MACHLETT DYNAMAX "60"**  
represents a refinement of the rotating anode X-ray source.





## a new capability for rotating anode x-ray tubes



The high energy capability of the Dynamax "60" permits fullest utilization of high power single and three phase equipment over a wide kilovoltage range. This capability extends to the fractional millimeter focal spot, and optional grid control, to give increased loads and exposure rates together with maximum rendition of detail.

For full information on the Dynamax "60" write The Machlett Laboratories, Inc., Springdale, Conn. 06879.

MACHLETT LABORATORIES, INC.



CLEAR CHOICE IN  
HYSTEOSALPINGOGRAPHY

WATER-SOLUBLE CONTRAST MEDIUM

**SALPIX<sup>®</sup>**

0.53 Gm Sodium Acetrizate and 0.23 Gm Polyvinylpyrrolidone per cc

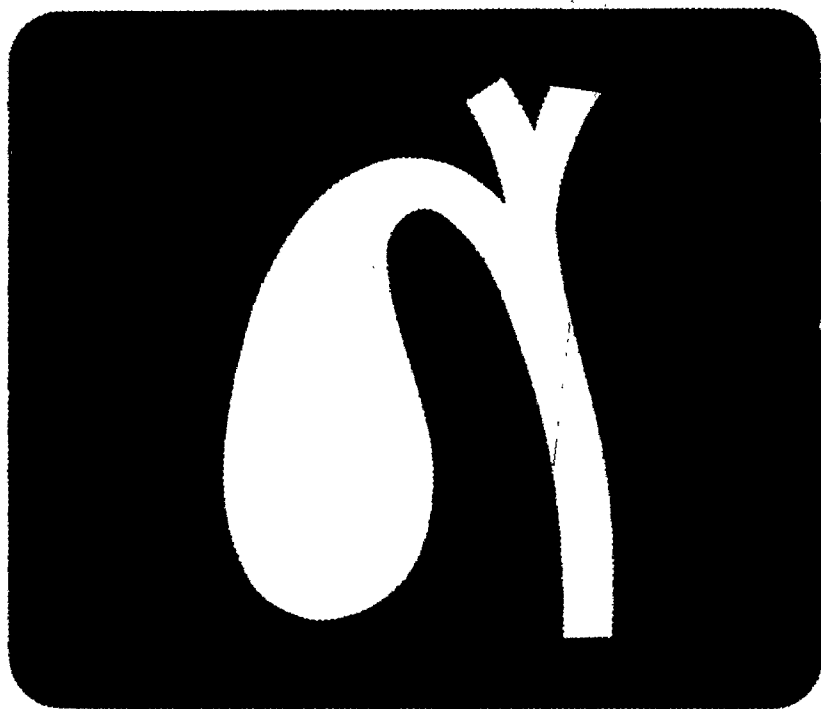
Fast, safe, accurate radiopaque visualization  
without irritation, residue, or pain



ORTHO PHARMACEUTICAL CORPORATION • RARITAN, NEW JERSEY

**CONTRAINDICATIONS**—Contraindications to hysterosalpingography include the presence of severe vaginal or cervical infections, existing or recent pelvic infection, marked cervical erosion or endocervicitis, and pregnancy. The procedure is contraindicated during the immediate pre- or post-menstrual phase. **SENSITIVITY**—If indicated in the patient's history, an intracutaneous skin test or sublingual absorption observation may be done with 0.1 cc SALPIX. **WARNING**—Not for intravenous use.

©CPC 1966



## **"Silent" gallbladders... speak clearly to radiologists**

Cholecystography consists of radiologic examination following the oral administration of a radiopaque agent (such as TELEPAQUE®) which is excreted by the liver into the bile, and is concentrated in the gallbladder. This concentration provides highly contrasted gallbladder shadows and often obtainable clear definition of the ducts. Evidence indicates cholecystography should be a routine addition to any gastrointestinal "work-up" you order for your patient, to help unmask "silent" gallstones or other gallbladder pathology.

The single most important attribute of any cholecystographic contrast agent is its accuracy in diagnosing biliary tract disease. From this standpoint, TELEPAQUE provides the highest degree of diagnostic accuracy, usually with only one dose, and one examination. Nonvisualization means gallbladder disease;

good visualization means a functioning gallbladder—and you can depend on this diagnosis.

But this is only one of the numerous precise radiodiagnostic procedures—many of them perfected during the last few years—now provided by radiologists who can be of invaluable assistance to you and your practice.

**Usual dosage of TELEPAQUE**—3 Gm. (6 tablets) at night after a light supper.

Tablets are supplied in envelopes of 6, in boxes of 5 and 25 envelopes.

**Side Effects** are usually mild, and may include nausea, vomiting, diarrhea, cramps, headache, dysuria and, rarely, skin rash, urticaria, pruritus, flushing.

**Contraindications** are acute nephritis, uremia, and gastrointestinal disorders which prevent absorption of the medium.

**Winthrop** PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS

WINTHROP LABORATORIES  
New York, N.Y. 10016

This advertisement—one in a series by Winthrop Laboratories as a service to radiology—is now appearing in those medical publications which are widely read by general practitioners and specialists. It is designed to remind them of the many valuable diagnostic services they can obtain from radiologists.



# with Telepaque® (iopanoic acid) visualization means a functioning gallbladder nonvisualization means gallbladder disease\*

## "Silent" and "Vague" gallbladders often present diagnostic problems

Diseased gallbladders can be clinically "silent" or "vague" with confusing signs and symptoms, particularly cholecystitis *without* cholelithiasis. Both require the highest degree of diagnostic accuracy. (Of course, those with classic, readily diagnosable symptoms do not present a problem.) Autopsies indicate that approximately eleven per cent of the population in the United States have gallstones!<sup>1</sup> While females in the "4F" category—*fair, fat, forty and fertile*—are the most frequent victims, the disease is also quite common in males. Delay in diagnosis with resultant delay in employing appropriate measures, according to one authority,<sup>2</sup> "are fraught with disastrous results for the patient."

## "High diagnostic accuracy makes Telepaque the contrast agent of choice"

This is why TELEPAQUE with its unexcelled record of accuracy of 98.3% to 100% in large scale studies, has long been the oral contrast agent of choice in cholecystography and cholangiography.

TELEPAQUE helps you to establish the *functional* status of the gallbladder based on the *concentration* of the medium in the gallbladder—rather than by a *passive* filling of the organ by the agent. This highly important diagnostic attribute has been widely demonstrated by the superb degree of contrast obtained with TELEPAQUE often with only a "one dose, one examination" routine, so that visualization means a functioning gallbladder, nonvisualization means gallbladder disease. *And you can depend on what your films show.*

FOR COMPLETE PRESCRIBING INFORMATION, CONSULT THE PACKAGE INSERT.

**Side Effects:** TELEPAQUE has low toxicity, and side effects are usually mild. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea is mild, consisting merely of a few loose stools, although in isolated cases it may be severe. Other side effects reported include dysuria (stinging or pain on micturition), headache and, on rare occasions, skin rash, urticaria, pruritus, and flushing.

**Contraindications:** Acute nephritis, uremia, and gastrointestinal disorders that prevent absorption of the medium.

**Supplied:** Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

1. Newman, H. F., and Northup, J. D.: *Internat. Abst. Surg.* 109:1, 1959.

2. Weiss, S. (New York Polyclinic Medical School and Hospital): *Clinical Lectures on the Gallbladder*, Yearbook Publishers, Chicago, 1944, pp. 137, 149, 229.

\*An extraordinary high degree of accuracy has been achieved with Telepaque in studies involving large series of patients; however, unusual causes of nonvisualization other than gallbladder disease also exist.

# Telepaque®

for precise oral cholecystography  
and cholangiography  
brand of iopanoic acid

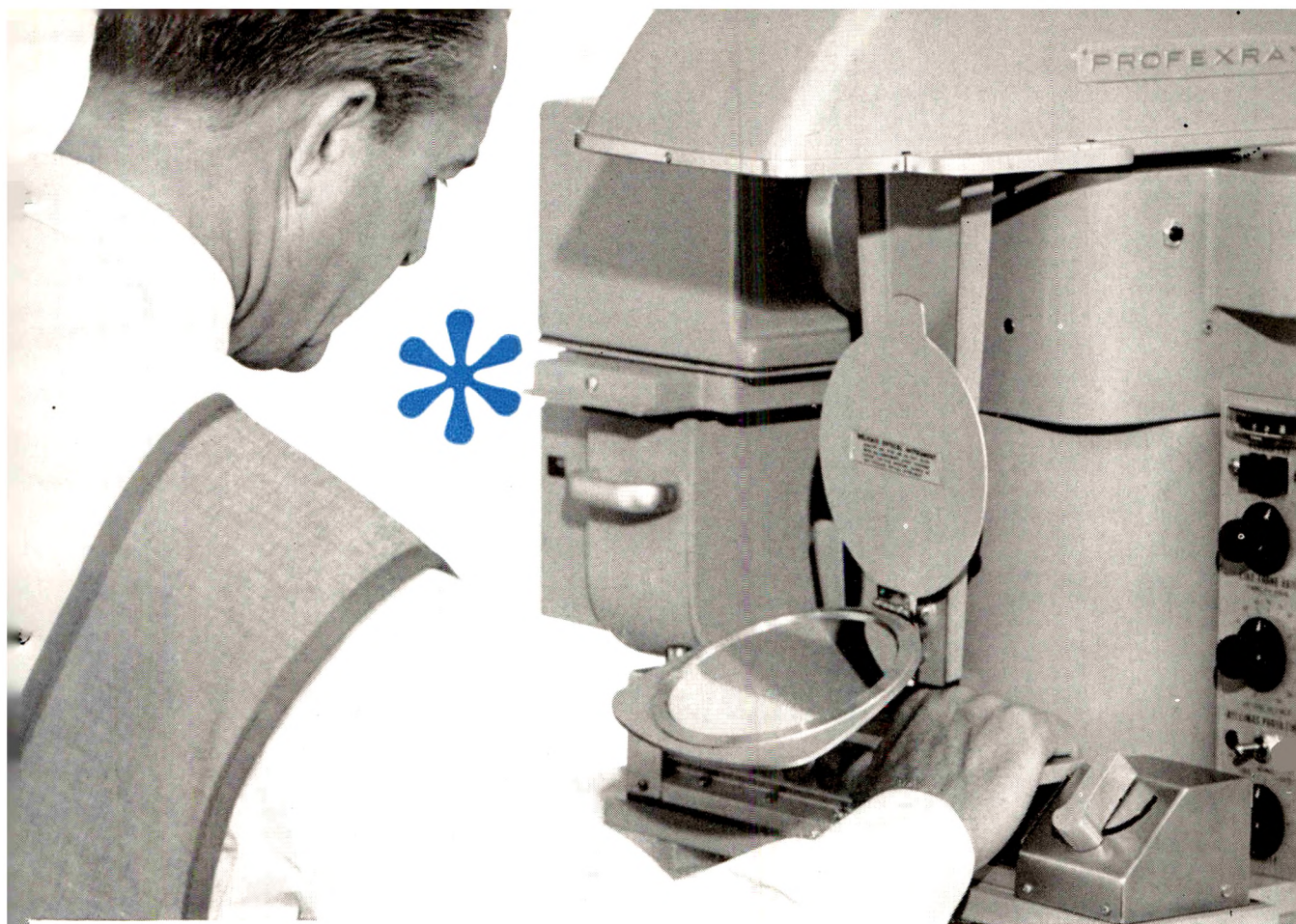


PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS

WINTHROP LABORATORIES  
New York, N.Y. 10016







## PROFEXRAY ADDS NEW DIMENSIONS TO THE CONCEPT OF **FLEXIBILITY**

### EMPEROR 90-360 TABLE—NEW

Heavy-duty, radiographic-fluoroscopic, 180° motor-driven table with tilt range from 90° Trendelenberg through horizontal to vertical.

#### **FLEXIBILITY**

1. A wide selection of spot film devices, image intensifiers, intensified image recorders, television systems, ciné systems or combinations of these systems has been developed for this table. (Pictured here with PIP-6 Image Intensifier and Intensified Image Recorder.)
2. A true island-type table. Unique design eliminates projections from table body by removing drive motor and transmission from rear of table—results in 360° access to patient.
3. Fluoroscopic and radiographic studies are made easy, using the manual-glide or motor-driven table top, which are available as options.
4. Three smooth motor drives are available: single-speed, dual-speed, or continuously variable speed.



### INTENSIFIED IMAGE RECORDER—NEW

Provides records (spot films) of the intensified fluoroscopic image with a degree of definition not obtainable with conventional spot films: (1) at intervals controlled by operator, (2) sequentially at rates up to six exposures per second

#### **FLEXIBILITY**

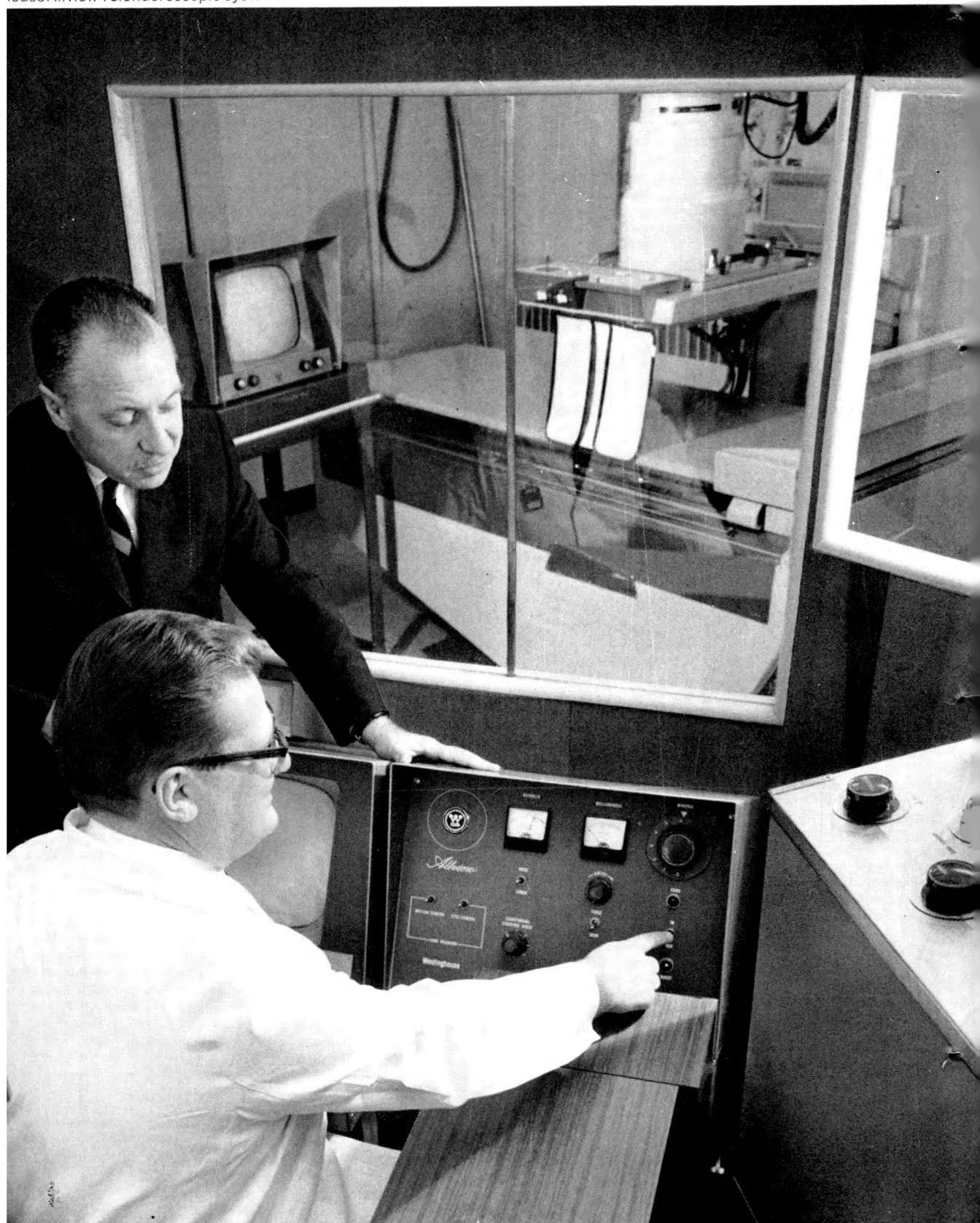
1. May be used with or without spot film device—significant advantages are to be gained when used *place of* spot film device.
2. Exposure factors to record the fluoroscopic image 1/10 to 1/20 of those used for regular spot film studies. These lower factors permit the use of a fractional-focal tube, resulting in films of unparalleled sharpness. Magnification studies become routine.
3. A complete range of lenses is available, and these easily interchanged. 90 mm and 70 mm magazines can be interchanged. Ciné and television cameras are available.
4. May be permanently installed on this table (Emperor 90-15), with associated options, without necessity of a cumbersome ceiling suspension system.

PROFEXRAY |



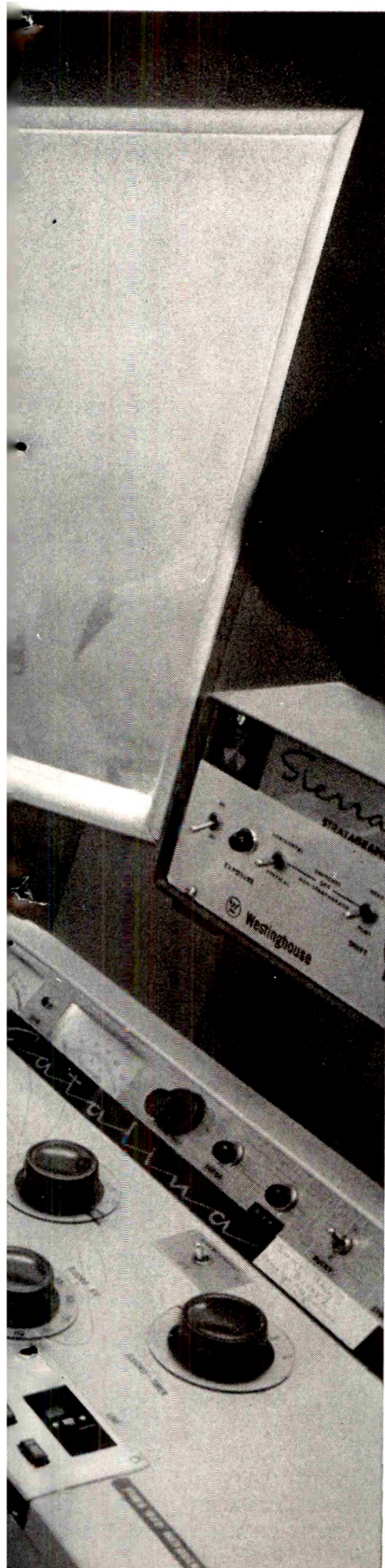
ard Goldberg, Chief Radiologist, and  
rk T. Seymour, Hospital Administra-  
ne Northwestern General Hospital,  
Ontario, Canada. The equipment:  
house Allview Telefluoroscopic System.

# The Westinghouse Professional





# Commitment to Radiology at work in Toronto



The versatile Westinghouse Allview Telefluoroscopic System—recently installed in the Northwestern General Hospital, Toronto, Canada—is used for all gastro-intestinal and special procedures.

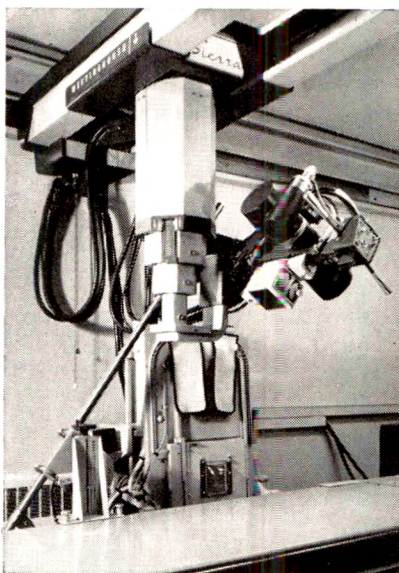
The Allview system of total fluoroscopy offers the freedom, convenience, and low-exposure advantages of remote control fluoroscopy...permits table-side or remote viewing on TV monitors. The Telecon vidicon camera is coupled with a 9" Image Amplifier that offers a brightness gain of more than 4000x—provides excellent resolution with as little as 0.1 footcandles.

A Westinghouse Stratograph is coupled with the Sierra Tube Stand for body-section radiography. Other equipment: Capri 90-15 motor-driven table and a Video Tape Recorder for examination review.

## What the Westinghouse Professional Commitment to Radiology offers you...

Facilities planning from simple layout to architectural plans and equipment specifications for a new hospital Radiological Suite... Design and installation of equipment to meet diagnostic specifications and case load requirements...Care of equipment and minimized downtime through our unique Preventive Maintenance Service Plan...Research and Development projects to meet the growing need for further advances, such as today's Image Amplifier—first introduced commercially by Westinghouse.

Ask your local Westinghouse X-Ray Representative to discuss how these services can help you, or for service and equipment information, write us. Westinghouse Electric Corporation, X-Ray Division, 2519 Wilkens Ave., Baltimore, Md. 21203.



The Westinghouse Stratograph permits body-section radiography, in addition to all other procedures possible with the versatile Allview system. The Stratograph offers such advantages as selective depth of cut indexed from 0 to 25 cm, selective distance of travel, selective thickness of cut from 2 cm to 6 cm in horizontal and vertical body-section radiography. J-08454



## You can be sure if it's Westinghouse



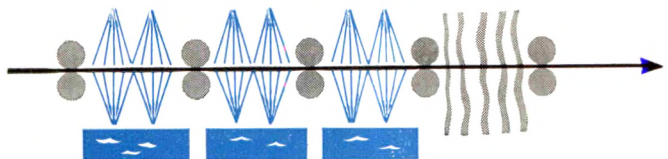
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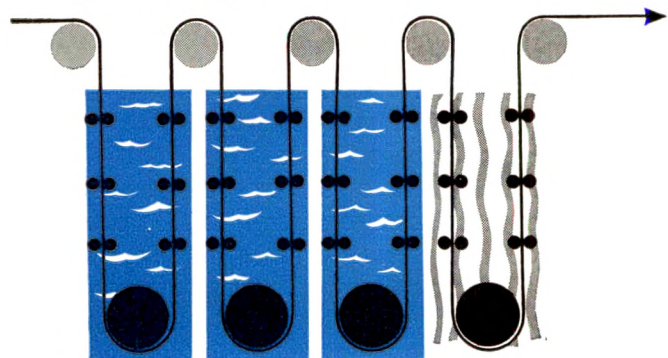
to here



this is a better way



than this



right?

**right!**

And that's the way films go through the processing cycle in our PIXAMATIC model III.

Straightline throughput, rather than tortuous meandering, zips them through at 49.5 inches per minute. Which means it can deliver a dry, ready-to-read 14" x 17" rapid-process type x-ray film in 69 seconds. Or an 8" x 10" in less than a minute (58.1 seconds to be exact).

Now if you're not in that much of a hurry, our other straightline PIXAMATIC (model II) will put any kind of film through at twelve inches a minute, delivering a dry 14" x 17" in 4.6 minutes. An 8" x 10" in 4.

You decide which is right for your kind of work, your traffic load.

With either PIXAMATIC you get these extra (unique) benefits . . .

**No abrasion artifacts.**

*Films ride flat on soft rollers, bathed on both sides by jet sprays.*

**No "lost" films.**

*All films always in plain sight by lifting cover. No possibility of jammed (and torn, maybe) films drowning in tank bottom.*

**Lower water consumption.**

*Needs only 1 gallon per minute (cuts water use by more than half: important in drought-stricken areas).*

**No daily cleaning.**

**Standby idling.**

*Film transport operates only while films are in the machine: saves wear and tear.*

**Easier to operate.**

**Makes sense, doesn't it?**

Your local Picker representative will be glad to fill you in on details. Have him in, or write us for fuller description and specifications

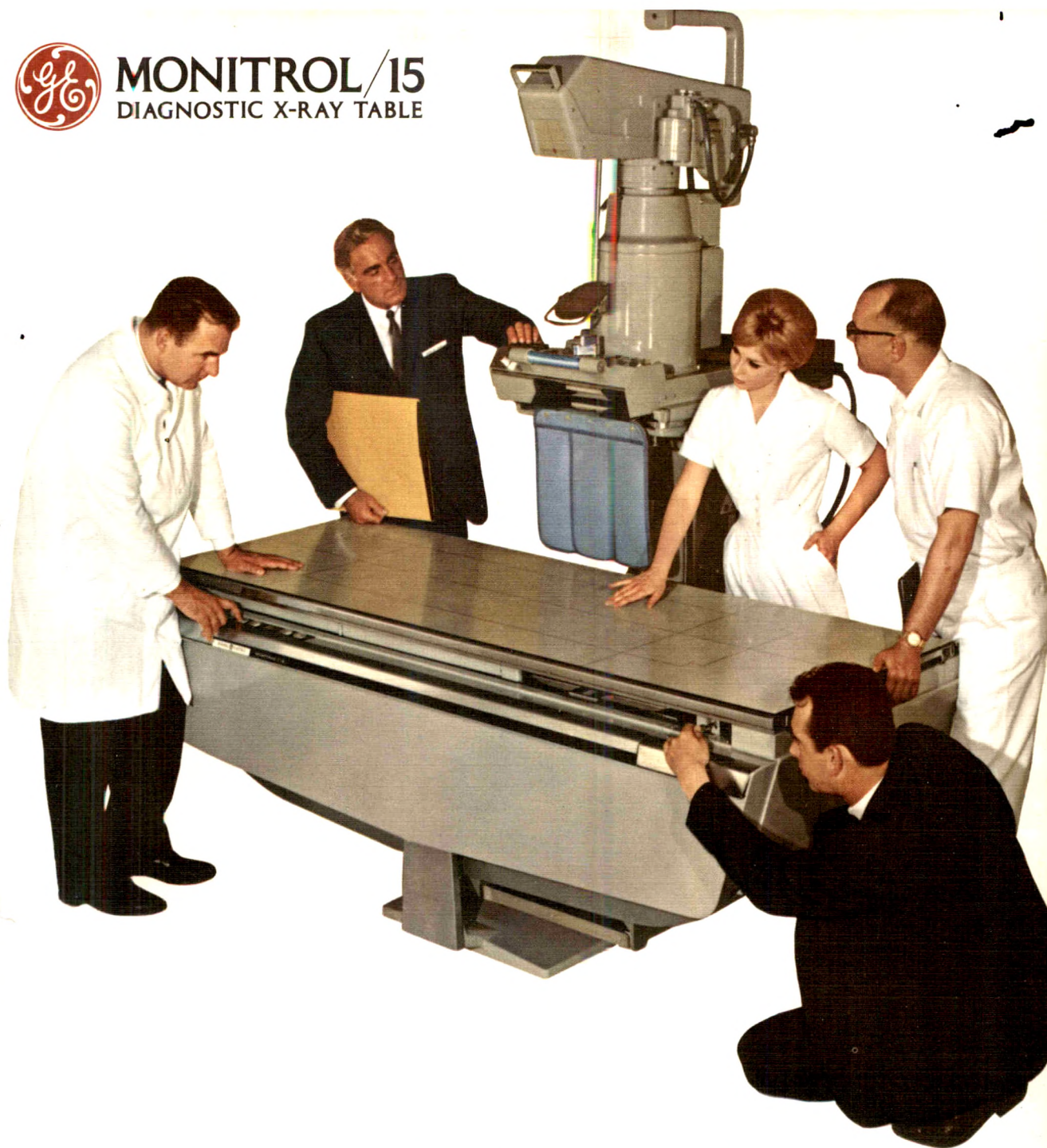


PICKER X-RAY CORPORATION  
White Plains, N.Y.





## MONITROL/15 DIAGNOSTIC X-RAY TABLE



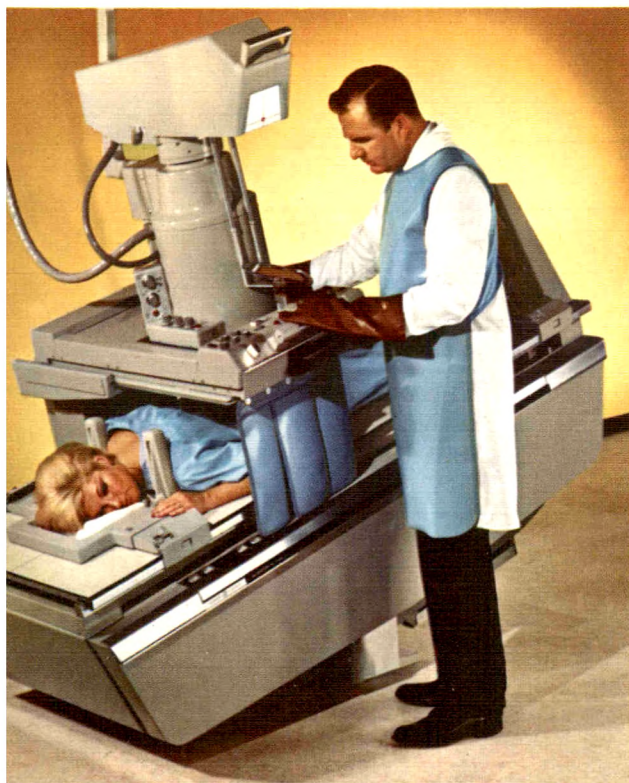
### ...invites critical inspection

**Monitrol/15**—that's the new name to remember when you're selecting a heavy-duty diagnostic x-ray table. Designed and built to perform *all* radiographic and fluoroscopic functions, this new 90-15° table contains operational features never offered before, by anyone. A true, uncluttered island design, speeded-up powered motions and true field collimation of the fluoroscopic beam are but a few. And General Electric has produced all this at a moderate price.



# MONITROL/15

The New 90-15° Table Built For Heavy-Duty Use



The new Monitrol/15 fills the void between light-weight 90-15° tables and heavy-duty 90-90° hospital tables. It's built to take around-the-clock use and is offered by General Electric at an easy-to-accept price.

Every newly-designed feature of the Monitrol/15 is aimed at easing the task of the radiologist. For instance—the fluoroscopic tower is lower and far lighter in weight to let you move it with minimum effort and park it faster. Result: a less fatigued radiologist at the end of every examination. Glide-back parking of the spot film device is standard and allows for easy patient positioning and fast table top clearance for radiography.

The Monitrol/15 accommodates the entire Fluoricon line of image intensifiers. The fluoroscopic beam is defined by the G-E True Field Collimator for improved quality spot film and fluoroscopic image. Collimator is controlled by a servo-drive system providing an automatic relationship between blade and control handle positions. Feature for feature, the Monitrol/15 offers more in patient comfort and operator convenience than any diagnostic table made today. It's a proud addition to the General Electric line of superior X-ray equipment.

*Progress Is Our Most Important Product*



## Radiologist-Designed Features for Total Diagnostic Performance

**Spot Filming**—Monitrol/15 accepts a choice of top loading, end loading spot film devices and the Fluoricon Pedestal. New low tower provides for spot film travel of 11-in. Distance from table top to underside of spot film device ranges 7-in. to 18-in.



**Controls**—Dual control is standard on the Monitrol/15. Controls are positioned for ease of access on both the table-front ledge and on the spot film device. The table-mounted switches are barium-proof.

**Power Positioning**—The moving table top with independent centering switch provides for a choice of table head-and-foot travel at installation—choose from 30-in. head/30-in. foot,





NOW...  
A ONE-STEP PROCEDURE FOR  
EFFICIENT CLEANSING OF THE COLON  
PRIOR TO RADIOGRAPHY



Flat plate of  
abdomen,  
taken after  
administration  
of X-Prep Powder.

Superior Visualization...Fewer Retakes  
with

***X-PREP***  
**POWDER**

(standardized senna concentrate)

Easy-To-Mix With Water

$\frac{3}{4}$  oz. can

(complete adult dose)

***X-PREP***  
**LIQUID**

(standardized extract of senna fruit)

Ready-To-Drink

2½ oz. bottle

(complete adult dose)

**SIMPLE, PALATABLE, SINGLE-DOSE PROCEDURE FOR GOOD BOWEL PREPARATION**

A reliable, predictably effective bowel evacuant that achieves efficient cleansing of the colon by virtually colon-specific peristaltic stimulation. Reduces or eliminates the need for preparatory enemas. Highly palatable and easy-to-take, X-PREP Powder/Liquid encourages better patient cooperation. All the patient has to do is drink the preparation between 2 and 4 P.M. on the day prior to radiography.

© COPYRIGHT 1966, GRAY PHARMACEUTICAL CO.

**GRAY PHARMACEUTICAL CO./YONKERS, NEW YORK**



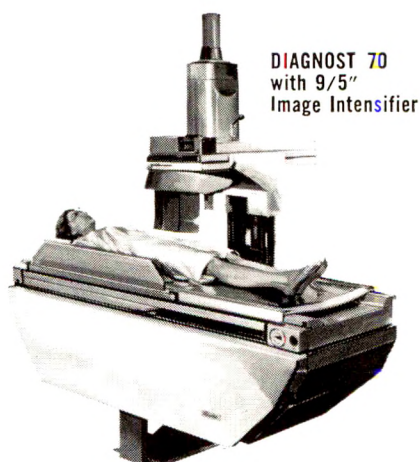
# Success hasn't spoiled NORELCO

Based on their quality radiographic and fluoroscopic performance, Norelco Image Intensifier Systems consistently lead the field. Compare results, compactness, stability, longevity and simplicity of operation with other systems. You will find no match for the Norelco 6" or 9/5" Intensifier Systems—because they were specifically designed for medical diagnostic use.

The high degree of contrast detail perceptibility and image clarity produced by Norelco Intensifier Systems is partly due to precision optical engineering. High-speed, large-aperture lenses (f 1:0.75)—deemed impossible a few years ago—are exclusive features with Norelco Intensifier Systems.

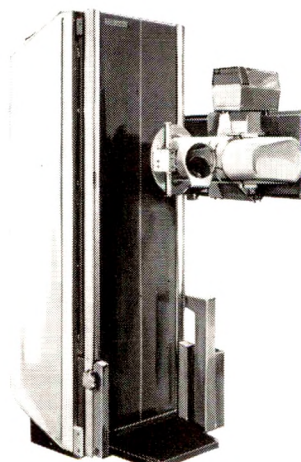
Two-channel distributors provide simultaneous viewing and recording: viewing via wide angle biocular mirror viewer or television, with cine or 70mm spotfilm recording. With the TV chain, video recorder and additional monitors for closed circuit TV are potential extensions of the system.

All Norelco Systems and components are completely integrated in design and manufacture. Norelco tables provide unusual operational facility with these systems—free from cumbersome ceiling suspensions.



DIAGNOST 70  
with 9/5"  
Image Intensifier

THE 9/5" IMAGE INTENSIFIER SYSTEM visualizes the 9" and 5" fields in a single intensifier tube. The 9/5" gives "panoramic" viewing of the 9" field, with immediate switch-over to the high resolution 5" center field, enlarged (1.8X) to the full diameter of the viewing screen.



DIAGNOST 50/6  
with 6"  
Image Intensifier

THE 6" IMAGE INTENSIFIER SYSTEM is clinically useful in such diverse applications as gastrointestinal examinations, heart catheterization, and fluoroscopic guidance in surgery. The compactness of the 6" Intensifier System is especially valuable for clinical investigation.

We are extremely proud of this technical achievement and would like to tell you about it in detail.

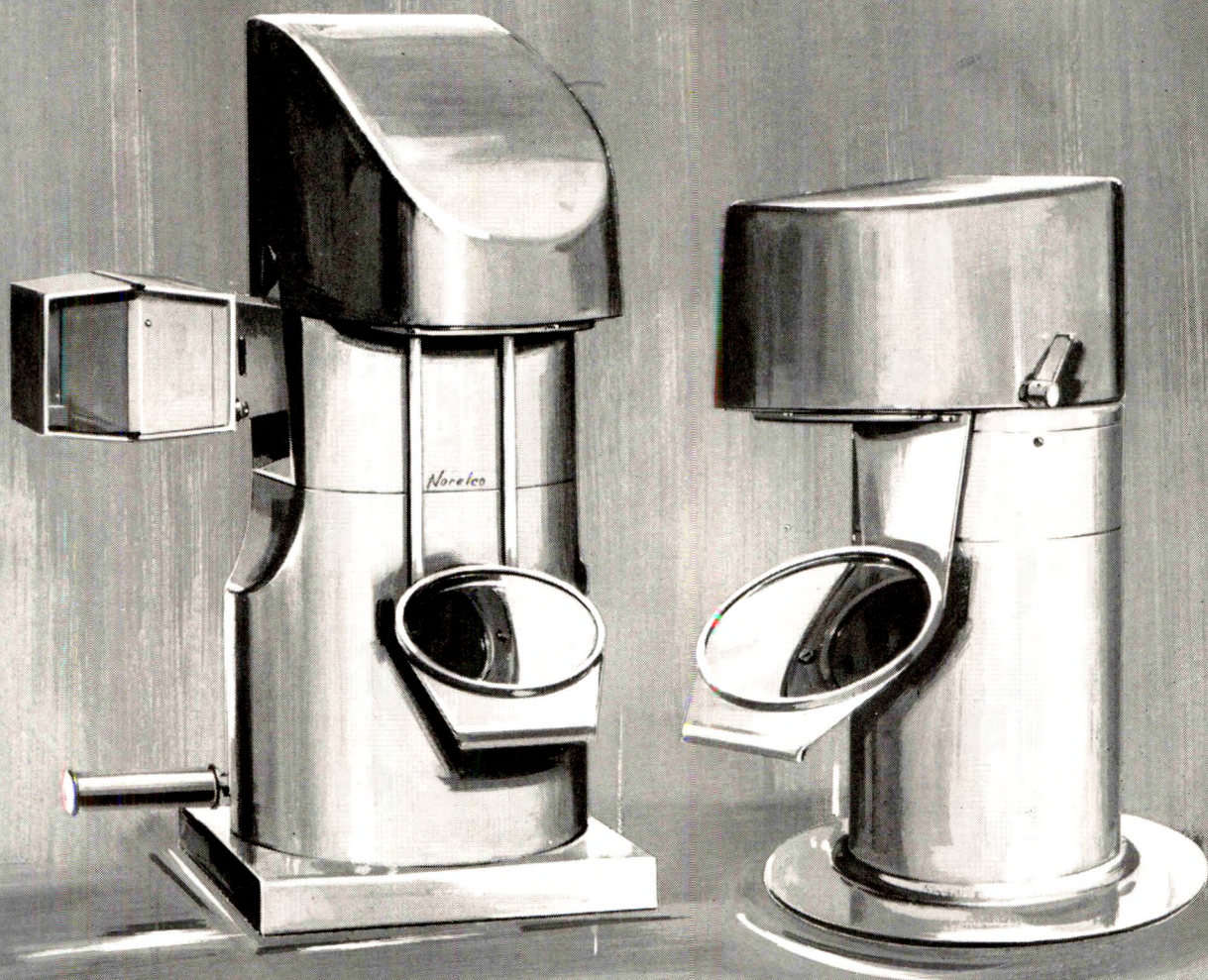
M-76



NORTH AMERICAN PHILIPS COMPANY, INC.  
Professional Products Division, 100 East 42nd St., New York, N.Y. 10017



# Image Intensifier Systems

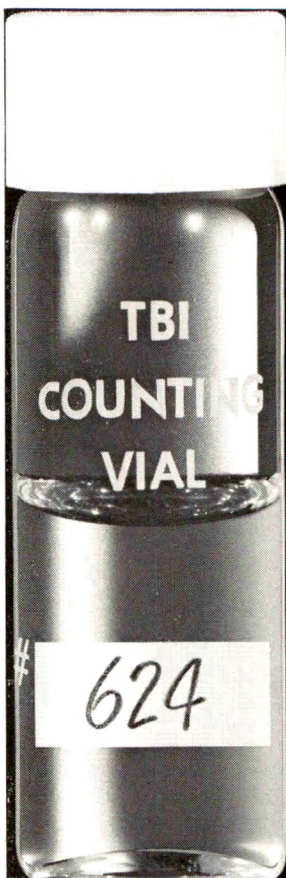


	9" (23cm)	9/5"	5" (12cm)	6"
Input Screen Diameter	9" (23cm)		5" (12cm)	6" (15cm)
Output Screen Diameter	13/16" (20mm)		13/16" (20mm)	9/16" (14.5mm)
Resolution (Central)	40 LP/inch		60 LP/inch	50 LP/inch
Resolution (Peripheral)	25 LP/inch		60 LP/inch	30 LP/inch
Intensification Factor (Average Gain)	5000		1500	5000
Conversion Factor*	36		12	36
Minimum Contrast	4%		4%	4%
Anode Voltage	25 kV		25 kV	25 kV
Viewing/Recording				

Biocular Mirror Viewer. High Speed Lens f1:0.75. Transistorized Medical TV Systems—Plumbicon†/Vidicon. Cine 16/35mm pulsed or non-pulsed. Spot-film Cameras 70mm Single Frame/High Speed.

\*Per ICRU recommendations in National Bureau of Standards Handbook. †Plumbicon is a registered trademark.





# TBI

*the T-3 test that*  
**PRACTICALLY TAKES  
CARE OF ITSELF**

## EASY

Technicians spend far less time with TBI... the *in vitro* thyroid function test that practically takes care of itself.

There's no stop watch timing. No critical temperature to maintain. And patient serum is counted only once. Total technician time is almost half that required of any other T-3 test.

## ACCURATE

Nuclear Consultants TBI, with its exclusive "Matched Control", offers unsurpassed accuracy for hyperthyroid, euthyroid and hypothyroid evaluation. In tests performed on over 2200 patients, the TBI test has been reported in agreement with 92% of the final clinical diagnoses of hyperthyroid patients; 89% of the euthyroid patients; and 96% of the hypothyroid patients.\*

\*Ref.: Scholer, J. F.,  
J. of Nuclear Med.,  
May '63, p. 192.

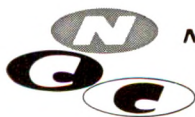
## DEPENDABLE

Primary binding sites are measured *directly*, eliminating the use of secondary sites and the uncertainty associated with indirect indication.

## COMPARE TBI

- New TBI buffer eliminates variables due to problems of serum handling, produces exceptional stability and reproducibility
- Unsurpassed accuracy for hyperthyroid — euthyroid — hypothyroid evaluation
- Timing and temperature are not critical
- Takes less technician time than any other T-3 test
- TBI low cost permits use as a screening test
- Available in kit and bulk form.

Write for further information and a copy of NCC's new booklet: *Thyro Binding Index.*



**NUCLEAR CONSULTANTS**

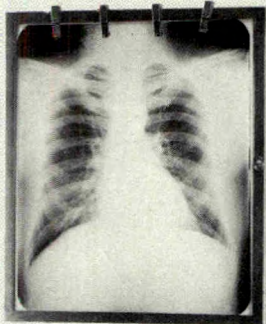
DIVISION OF MALLINCKRODT CHEMICAL WORKS

Box 6172 Lambert Field, St. Louis, Missouri 63145

ATLANTA • CHICAGO • CLEVELAND • LOS ANGELES  
MIAMI • NEW YORK • SAN FRANCISCO • WASHINGTON, D. C.



# R<sub>x</sub> for good radiographs



**The right  
film.**

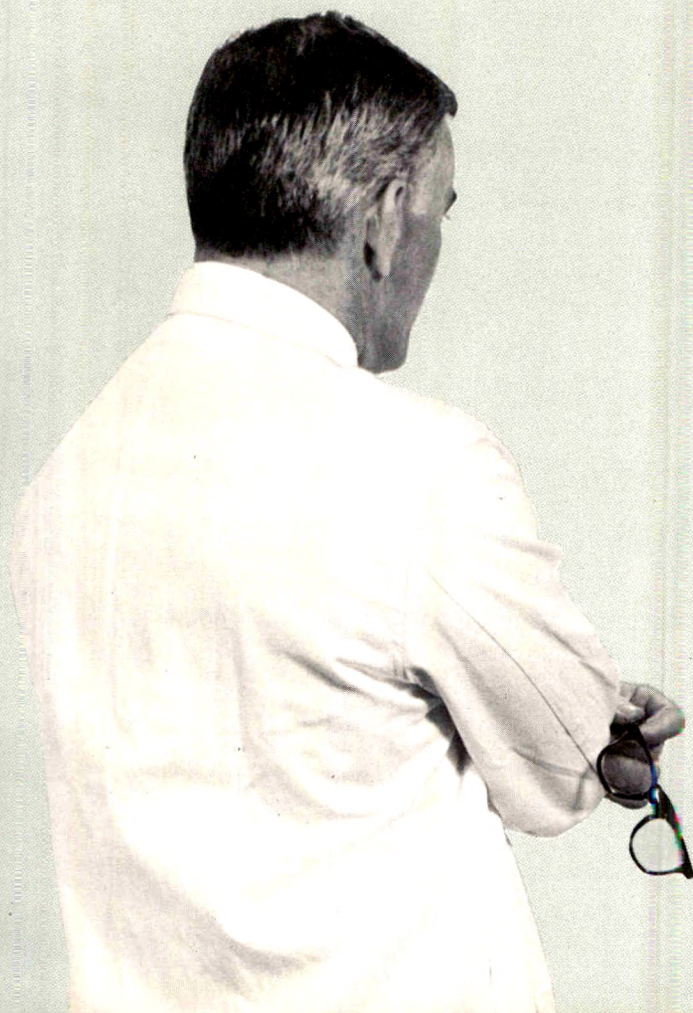
**The right  
screen.**

The right film for most radiographic investigations is one which offers high contrast, medium speed, fine grain and very low fog level. Ilford Standard is that film—at a cost appreciably lower than for other films of comparable quality. *Standard* is compatible with the new “half-cycle” or “double-capacity” automatic processing techniques.

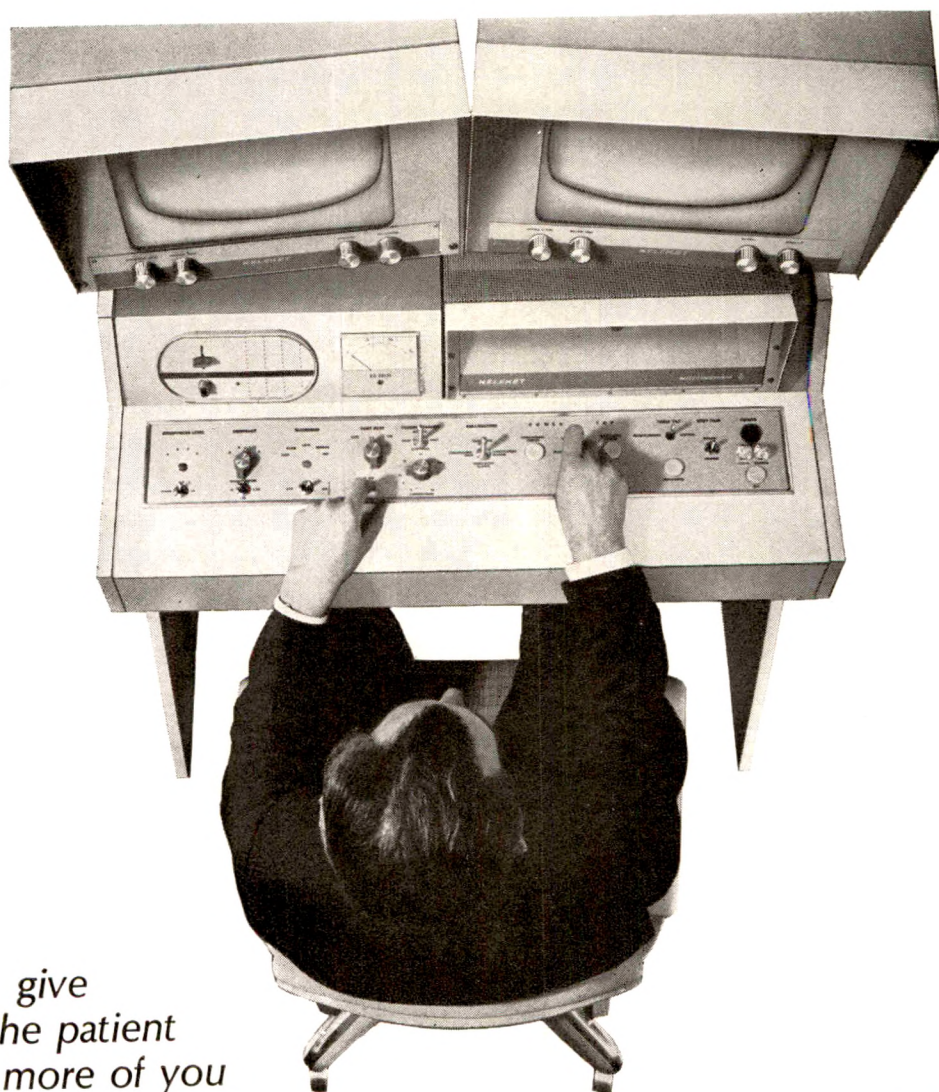
The right screen must be fast, with outstanding definition—and Ilford Fast Tungstate Intensifying Screens are the ultimate in the field; their definition is equal to that of most screens with half the speed. Put these two fine products in the hands of a skilled technician and you will have gone a long way toward improving your radiographs.

**ILFORD INC.**

37 West 65th Street  
New York, N. Y. 10023







give  
the patient  
more of you

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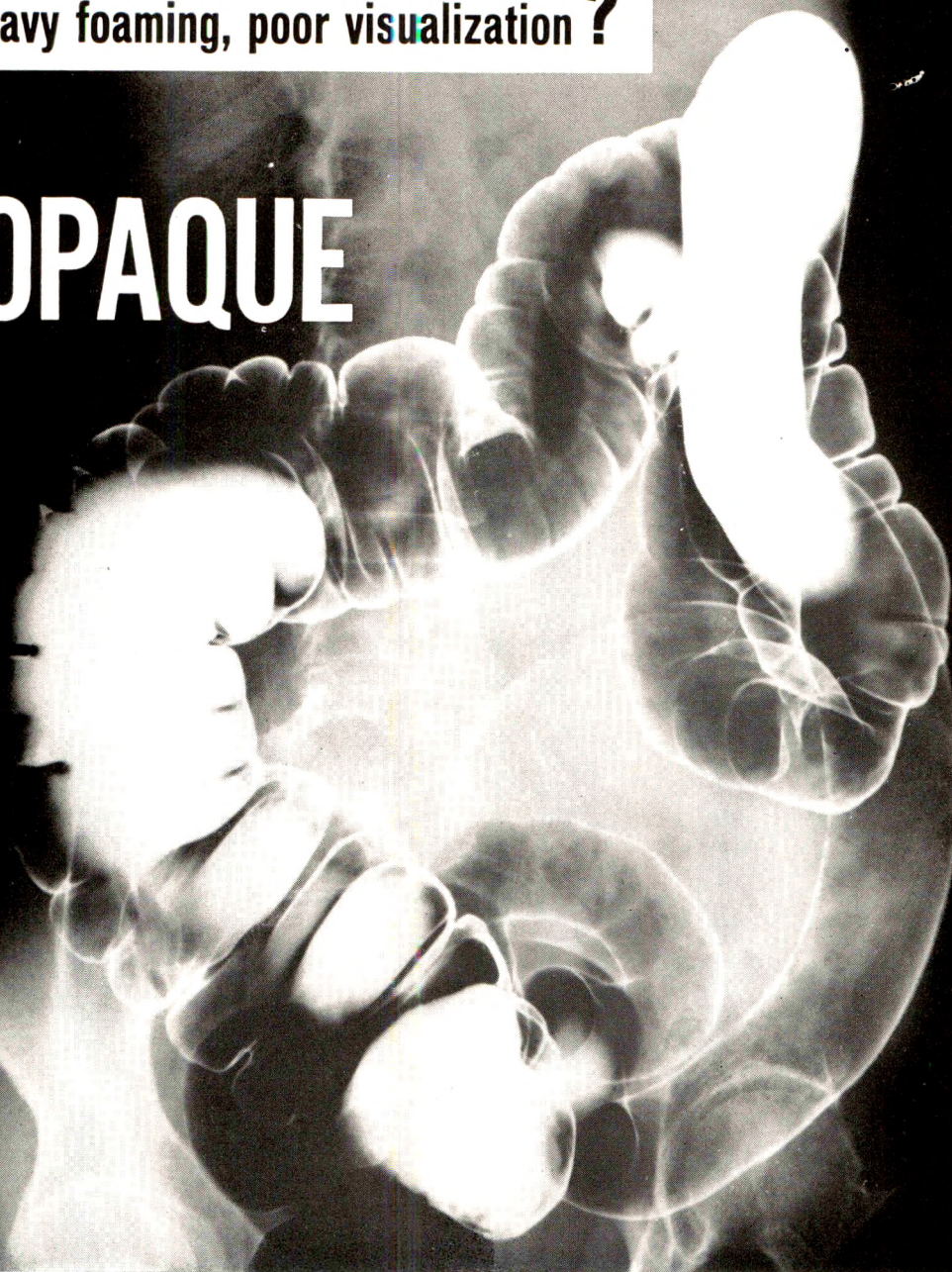
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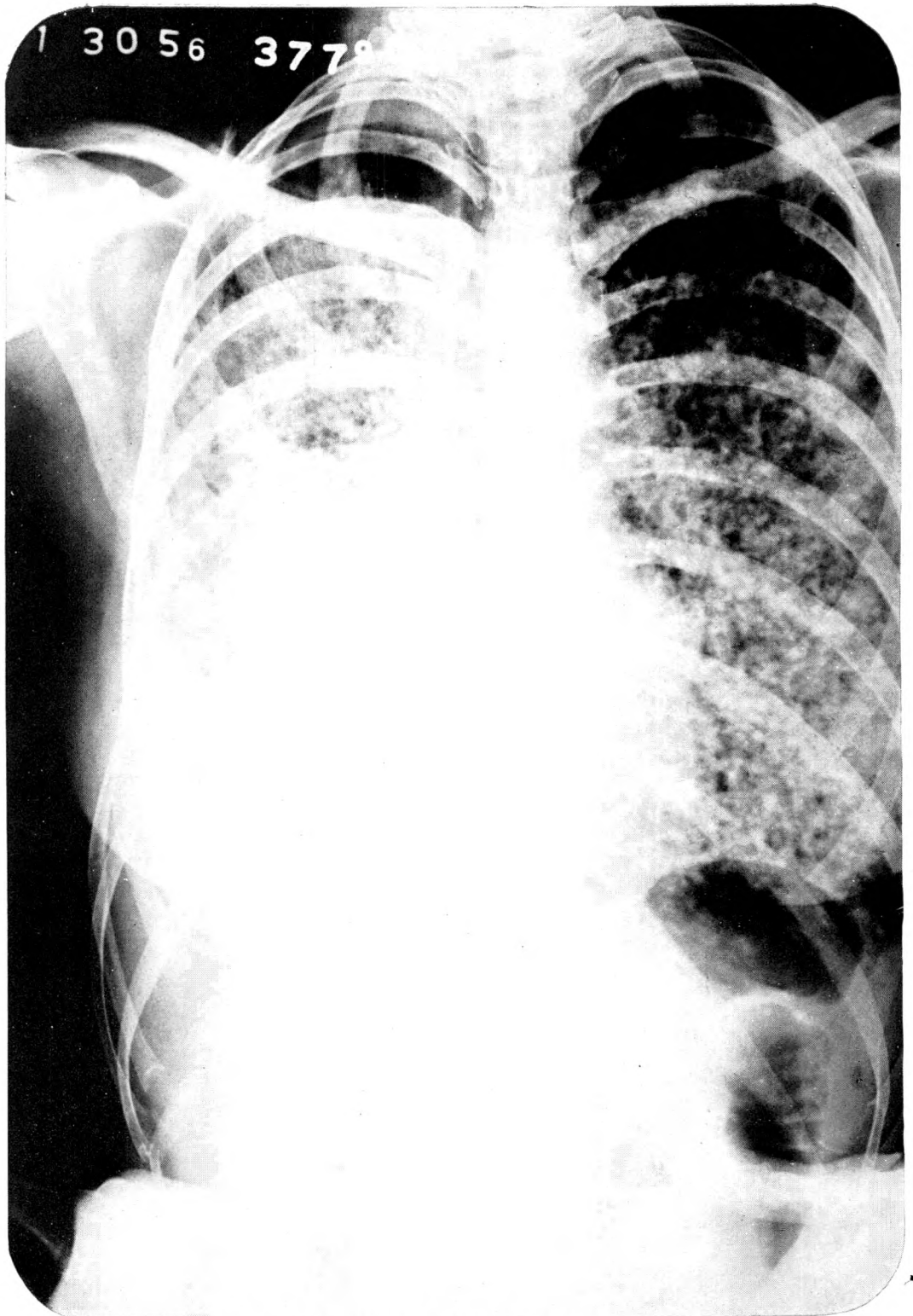
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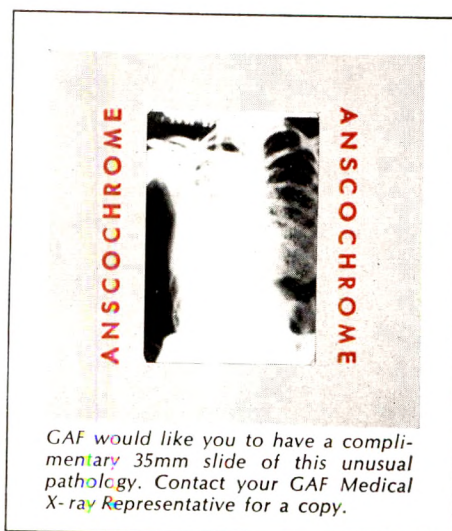
A 39-year-old woman had been well until Thanksgiving Day when she complained of shortness of breath. It disappeared—recurred on Christmas Day—and disappeared again. Thirty days later she was admitted with continuous and marked dyspnea that had appeared three days before admission. She had lost 20 pounds in the previous six months.

Physical examination revealed dullness and vocal fremitus with some wheezes and diminished or absent breath sounds over the right base posteriorly. Just above this area there was definite bronchial breathing.

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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE



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## SKULL CHANGES ASSOCIATED WITH INTRACRANIAL ARTERIOVENOUS MALFORMATIONS

By CALVIN L. RUMBAUGH, M.D.,\* and D. GORDON POTTS, M.D.†  
ST. LOUIS, MISSOURI

THE first arteriovenous malformation of the brain was reported by Steinheil in 1895, 138 years after William Hunter's original description of an arteriovenous aneurysm in the arm. In 1928 Dandy<sup>2</sup> described 8 of his own cases previously reported in the literature. Because angiography was not performed on these cases, he had to rely on the clinical findings, skull roentgenography, and ventriculography for their diagnosis. He noted calcification in an arteriovenous malformation, prominent vascular markings in the cranial vault, and a defect in the occipital bone caused by a large vein passing through the vault. In addition, he saw, at operation, thinning of the skull from its inner aspect caused by a superficial malformation, although this change was apparently not recognized in the roentgenograms. Dandy also noted that arteriovenous malformations of the brain could displace the ventricular system and simulate a tumor at ventriculography, and, in some cases, he saw enlargement of the ventricle adjacent to a malformation which

he considered to be the result of an old hemorrhage.

The diagnosis of an intracranial arteriovenous anomaly can almost always be made by angiography. A few cases have been reported by Kamrin and Buchsbaum<sup>3</sup> where angiography failed to show a large malformation and in such cases the malformation may show the angiographic appearance of a mass lesion without abnormal vessels.

Many cases present with a subarachnoid hemorrhage and often also a bruit which will suggest the diagnosis and indicate the need for angiography. However, in a number of cases the clinical findings are non-specific and there is no suspicion of an arteriovenous malformation at the time that roentgenograms of the skull are taken. In these cases it may be possible to diagnose, or at least suspect, the lesion from changes on plain roentgenograms.

### MATERIAL

Sixty-one cases of intracranial arteriovenous malformation of the brain demon-

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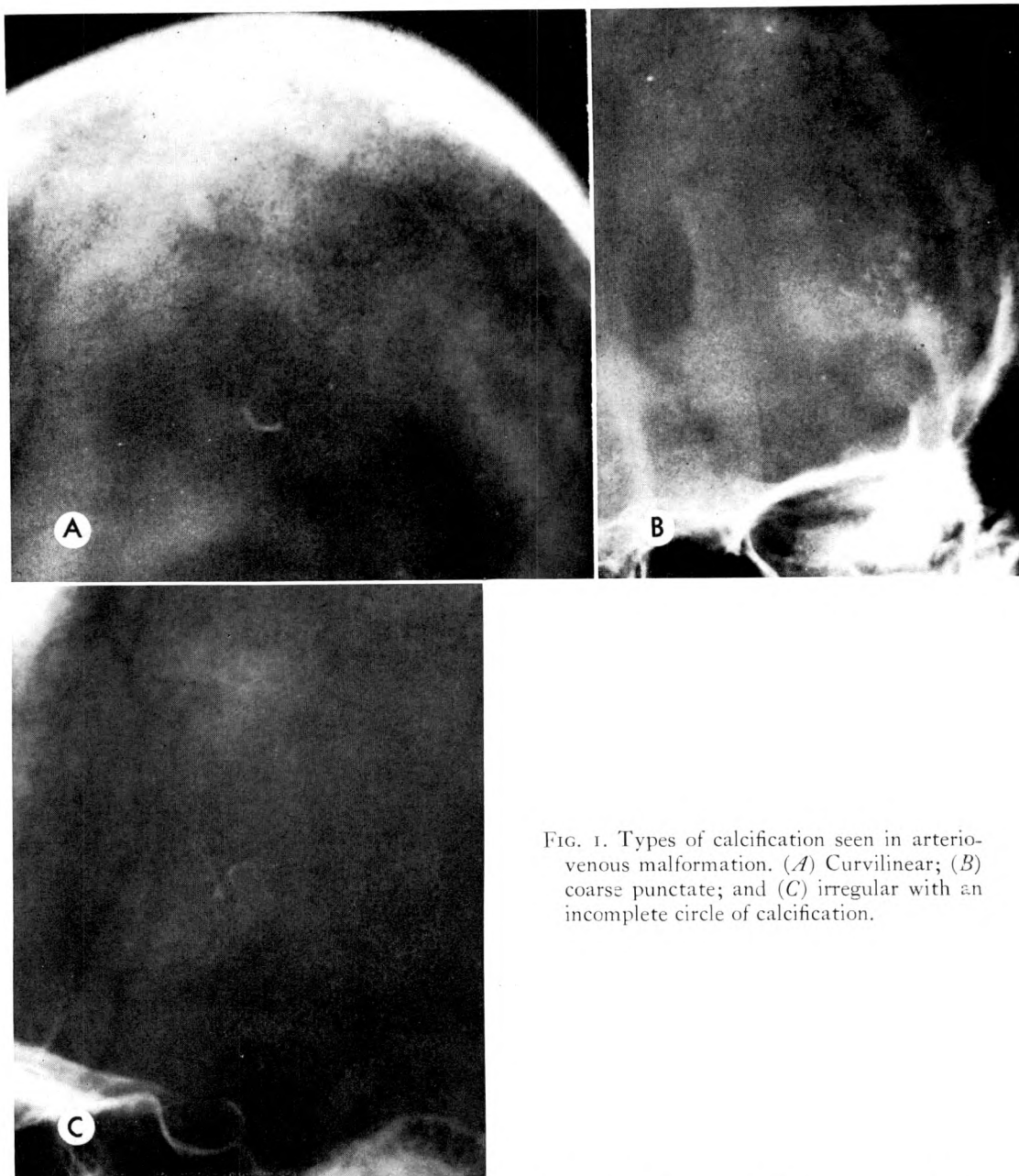


FIG. 1. Types of calcification seen in arteriovenous malformation. (A) Curvilinear; (B) coarse punctate; and (C) irregular with an incomplete circle of calcification.

strated by angiography were reviewed to determine the changes present in the plain skull roentgenograms, and these were correlated with the angiographic findings. These patients included 32 males and 29 females. All of the cases were admitted to the Neurological Institute, New York, and had simultaneous biplane angiography with at least 2 films per second in the arterial phase, and in some case up to 6 films per second.

The technique of the roentgenologic investigation has been described elsewhere by Pool and Potts.<sup>9</sup> Malformations associated with aneurysmal dilatation of the vein of Galen are not included in this series.

#### CLINICAL FEATURES

The age of the patients ranged from 11 to 67 years at the time that the diagnosis was made. Thirty-one (51 per cent) presented



with one or more subarachnoid hemorrhages and in 14 (23 per cent) a bruit was detected. Twelve (20 per cent) presented with focal or generalized seizures.

#### ABNORMAL INTRACRANIAL CALCIFICATION

Paterson and McKissock<sup>8</sup> found calcification in the malformation in 24 of 101 cases which had skull roentgenograms. In this series 18 (29.5 per cent) showed calcification which was linear, curvilinear, in the form of parallel lines, punctate, circular or irregular (Fig. 1, *A*, *B* and *C*). In many cases the calcification was similar to that seen in aneurysms or tumors.

#### DISPLACED PINEAL GLAND OR CHOROID PLEXUS

Eight (14 per cent) showed displacement of the calcified pineal gland or choroid plexus. This was usually due to the presence of an intracerebral hematoma, but occa-

sionally was seen when a large malformation occupied considerable space.

#### EVIDENCE OF ENLARGED INTERNAL CAROTID ARTERY

Radberg<sup>10</sup> has discussed the normal appearance of the carotid sulcus adjacent to the sphenoid sinus as seen in various projections. When the internal carotid artery enlarges, its sulcus becomes deeper and it extends farther forward in relation to the anterior wall of the sella turcica (Fig. 2, *A-D*). Frontal tomograms of the sella turcica may show abnormally deep carotid grooves (Fig. 3). The anterior clinoid processes tend to become elongated. Occasionally, the gross bilateral increase in the size of the internal carotid arteries may cause an appearance which simulates the enlargement of the sella turcica due to an intrasellar tumor (Fig. 4, *A-D*). However, the normal carotid sulcus shows consider-

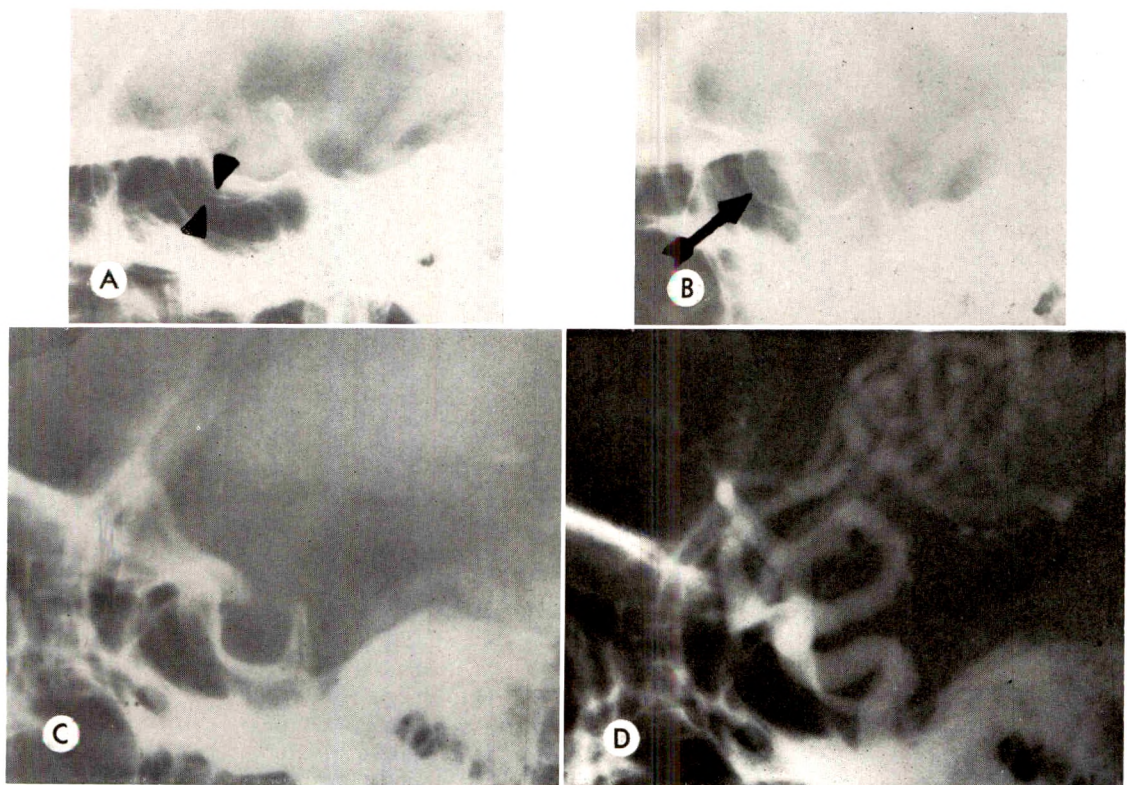


FIG. 2. Deep carotid sulcus extending farther anteriorly than usual. (*A*) Enlarged carotid sulci on both sides (arrows). (*B*) Enlarged carotid arteries on both sides but carotid sulci superimposed (arrow). (*C*) Skull roentgenogram and (*D*) arteriogram showing enlarged carotid artery and its prominent sulcus.



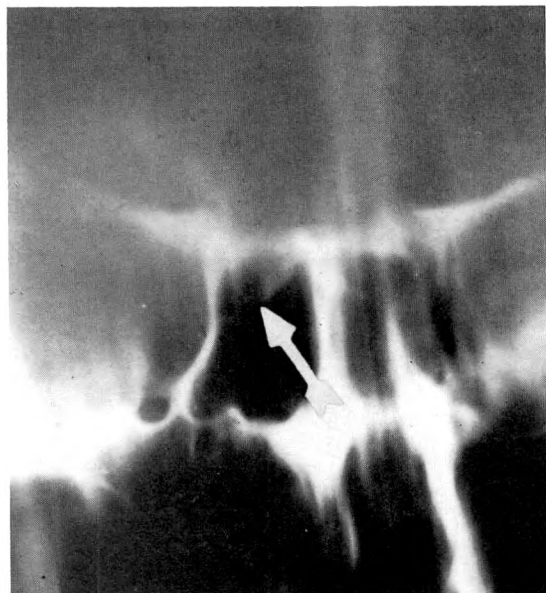


FIG. 3. Deep carotid groove (arrow) shown by frontal tomography.



able variation and only rather gross enlargement of the internal carotid artery will cause changes that can be recognized with any degree of confidence. In many cases, there will only be a suspicion that the internal carotid artery is enlarged from the appearance of the carotid sulcus and one will have to look elsewhere for confirmatory evidence.

As it lies in the petrous temporal bone, the outer diameter of the internal carotid artery may sometimes be demonstrated

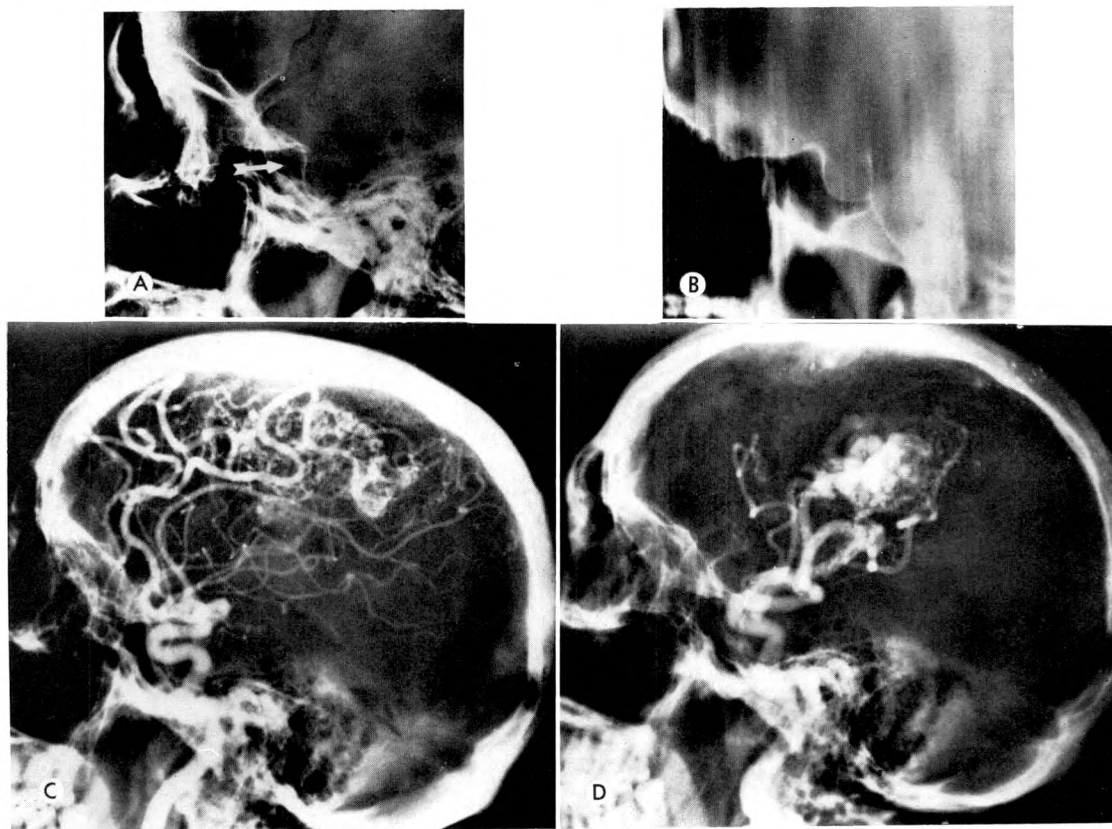


FIG. 4. Enlargement of the sella turcica caused by marked enlargement of the internal carotid arteries. (A) Lateral roentgenogram of sella turcica. The sella turcica has an anteroposterior measurement of 18 mm. and a depth of 17 mm. The carotid sulci extend still farther anteriorly (arrow). (B) Lateral tomogram of sella turcica. (C) Left carotid arteriogram. (D) Right carotid arteriogram. The malformation involved the right hemisphere. Note thick cranial vault. (Courtesy of Dr. Ernest Wood. This case was not included in the series.)



quite clearly. The artery enters the carotid canal in the petrous temporal bone, passing rostrally for a short distance (Fig. 5), and then forward and medially for a longer distance to reach the region of the foramen lacerum medium. This latter portion of the vessel may be more or less horizontal, or it may pass somewhat caudally as it runs forward and medially. The vertical portion of its course may not be seen in the base view and it may even be quite difficult to demonstrate by tomography of the base of the skull because it is so short, particularly its medial margin.

The more medial part of its course in the petrous temporal bone may be difficult to demonstrate in routine studies because of its obliquity and since it is not demarcated by compact bone. It is usually very clearly seen if it is viewed along its long axis (Fig. 6, *A* and *B*), but because of the variable course of this portion of the vessel, it may be difficult to obtain the correct view to show the canal in this way. It is probable that, with careful television fluoroscopy, the correct projection could be obtained in almost every case.

As the internal carotid artery enlarges in

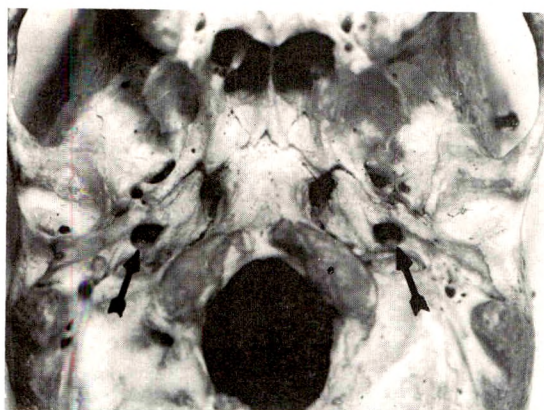


FIG. 5. View of inferior aspect of skull showing carotid canals (arrows).

diameter, it usually also increases in length. When this occurs, it may cause an abnormally prominent groove on the lateral aspect of the body of the sphenoid, adjacent to the apex of the petrous temporal bone (Fig. 7). Ten cases in this series showed changes in the skull roentgenograms which were felt to indicate enlargement of the internal carotid artery. These cases all had very large arteriovenous malformations and in most cases the internal carotid arteries on both sides were grossly enlarged.

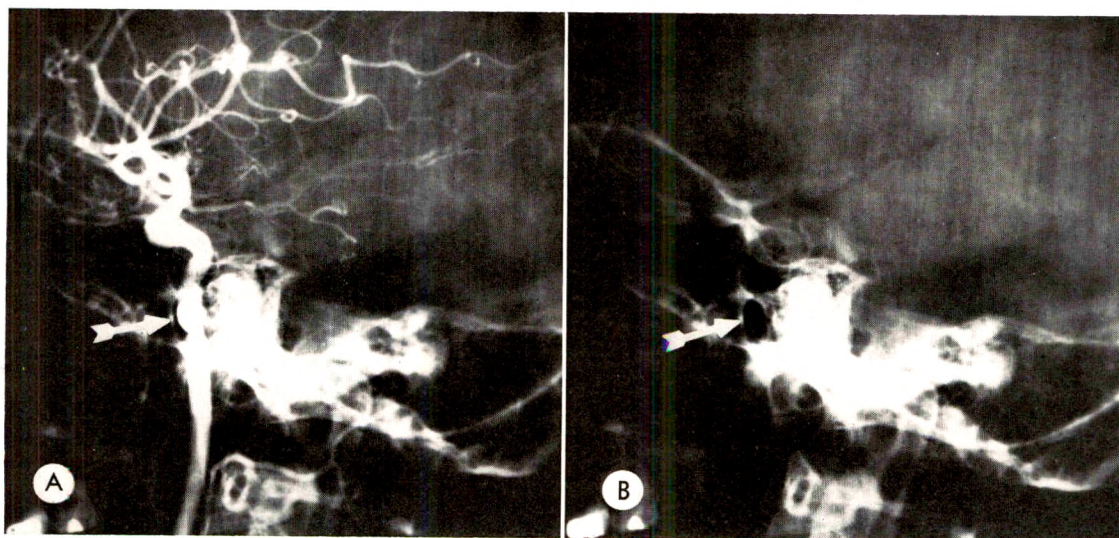


FIG. 6. Axial studies of medial portion of bony carotid canal within the petrous temporal bone in rotated lateral view. (*A*) Arteriogram showing axial view of the internal carotid artery as it passes forward and medially in the petrous temporal bone (arrow). (*B*) Plain roentgenogram in the position of angiography showing carotid canal (arrow). The head is rotated 30° away from the side of injection.



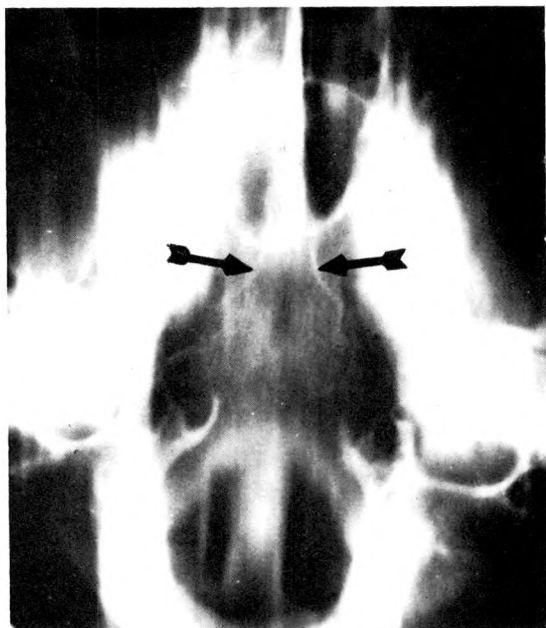


FIG. 7. Tomogram of base of skull showing prominent grooves on the lateral aspects of the body of the sphenoid (arrows). The jugular fossae are also enlarged.

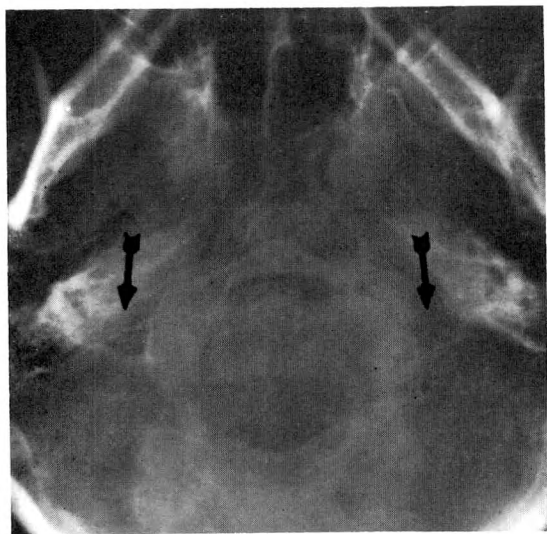


FIG. 8. Base view of the skull showing enlarged foramina of the vertebral arteries in the first cervical vertebra (arrows).

#### EVIDENCE OF ENLARGED VERTEBRAL ARTERIES

Enlargement of the foramina of the vertebral arteries in the first cervical vertebra could be detected in the base view (Fig. 8),

or in lateral views where an abnormally deep groove on the upper aspect of the posterior arch of the first cervical vertebra could be seen (Fig. 9). Occasionally, the vertebral artery was encircled by bone in this region, and the foramen formed indicated the presence of a large vertebral artery (Fig. 10, *A* and *B*).

Although Lindblom<sup>5</sup> felt that the size of the foramen transversarium of the first cervical vertebra was not as good an index of the vertebral artery size as the foramen transversarium of the second cervical vertebra, it seemed to be of some value in the present series.

#### VESSELS PENETRATING CRANIAL VAULT

When the malformation was supplied by external carotid vessels in the scalp or when veins draining the malformation passed through the cranial vault, the lucencies where the vessels penetrated the cranial vault could sometimes be seen. These most commonly caused small round areas of reduced density and they usually occurred in relation to the more superficial vascular malformations (Fig. 11). These perforating channels may be difficult to recognize if they are very small, particularly if they are not seen end-on in any view. However, they are of some importance as they may provide the only evidence on plain roentgenograms of the position of a superficial malformation. Only 2 cases in the present series showed evidence of these perforating chan-

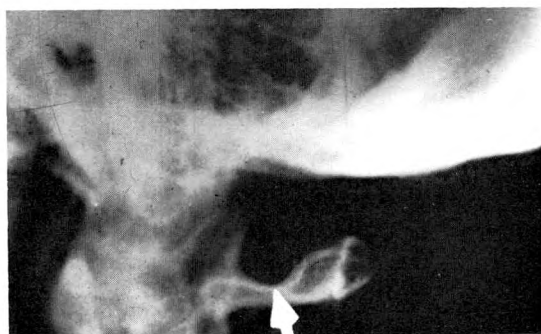


FIG. 9. Prominent groove on the upper aspect of the arch of the first cervical vertebra (arrow) caused by a large vertebral artery.



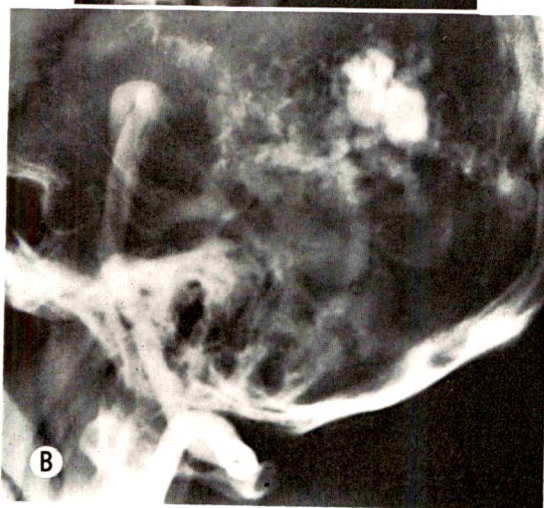
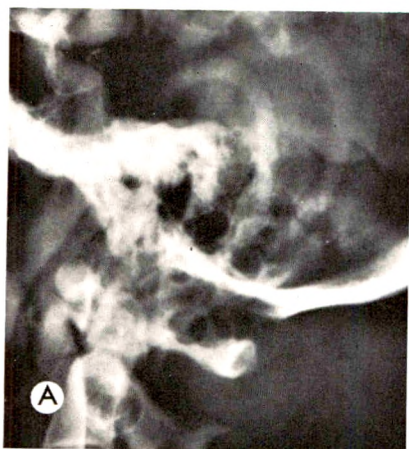


FIG. 10. (A) Complete rings of bone surrounding the vertebral arteries as they lie above the posterior arch of the first cervical vertebra. Both vertebral arteries were enlarged. (B) Arteriogram showing one of the large vertebral arteries.

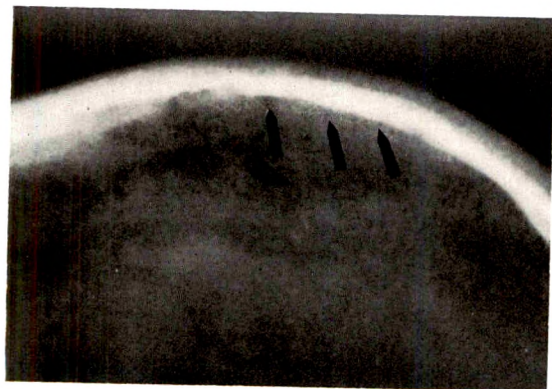


FIG. 11. Small channels (arrows) caused by vessels passing through the cranial vault over the surface of a superficial malformation.

nels in the skull roentgenograms. Occasional, perforating channels may be seen in a normal skull, but a group of these channels usually indicates the presence of an arteriovenous malformation. Other cases were shown angiographically to have these channels, but they could not be recognized in the plain skull roentgenograms.

Two cases showed evidence of enlarged occipital emissary veins similar to those described by Chynn,<sup>1</sup> presumably caused by increased venous pressure at the point of junction of the 2 lateral sinuses (Fig. 12).

#### CHANGES CAUSED BY ENLARGED VEINS AND VENOUS SINUSES

When a large arteriovenous shunt is present, the grooves in the skull caused by veins or venous sinuses may become enlarged. Because these vary so much even in normal patients, only rather gross enlargement is diagnostically helpful (Fig. 13). The jugular foramina may also be enlarged (Fig. 14) and prominent venous markings may be seen on the inner aspect of the cranial vault.

#### ENLARGED ARTERIAL MARKINGS ON INNER ASPECT OF CRANIAL VAULT

When enlarged meningeal arteries supply the malformation, large (and often tortu-



FIG. 12. Enlarged occipital emissary veins associated with a large vascular malformation.



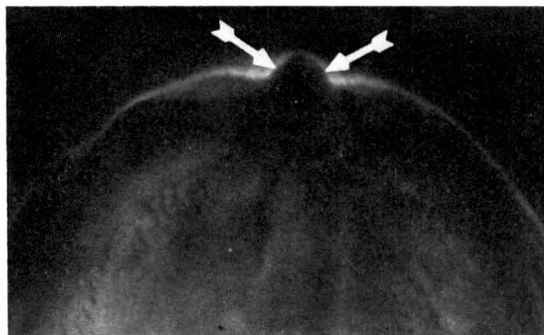


FIG. 13. Large groove on inner aspect of skull caused by enlargement of the sagittal sinus (arrows).

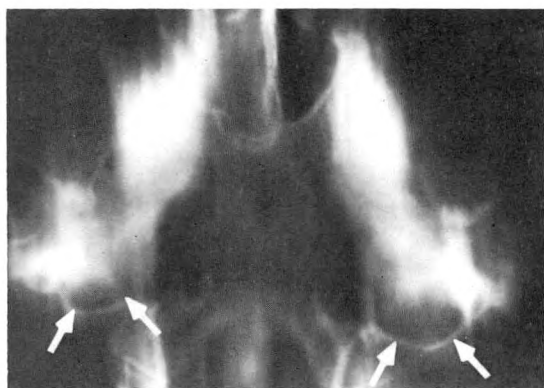


FIG. 14. Large jugular foramina (arrows) shown by tomography of the base of the skull

tous) grooves may be seen on the inner aspect of the cranial vault (Fig. 15). Seven cases in the present series showed prominent meningeal arterial grooves. When the middle meningeal artery was enlarged, the foramen spinosum was also enlarged. Because of the frequent asymmetry of these foramina on the 2 sides of a normal skull, a foramen spinosum should only be presumed to be enlarged if large arterial markings on the inner aspect of the skull can also be recognized. A prominent groove may be seen extending laterally from the large foramen spinosum in the base view.

#### THINNING OF THE CRANIAL VAULT

Two cases showed thinning of the cranial vault from its inner aspect. This occurred when the malformations or grossly enlarged veins were on the surface of the

hemisphere adjacent to the inner table of the skull (Fig. 16).

#### THICKENING OF THE CRANIAL VAULT

In 3 cases there was some thickening of the cranial vault and a reduction of the size of the hemicranium on the side of a vascular malformation. The digital markings on the inner table were also reduced on the side of the lesion.

Three cases with large arteriovenous shunts showed a generalized thickening of the cranial vault (Fig. 17, *A* and *B*). Unexplained thickening of the cranial vault is sometimes seen in otherwise normal pa-



FIG. 15. Enlarged meningeal vascular marking (arrows). There was some calcification in this large parietal malformation.

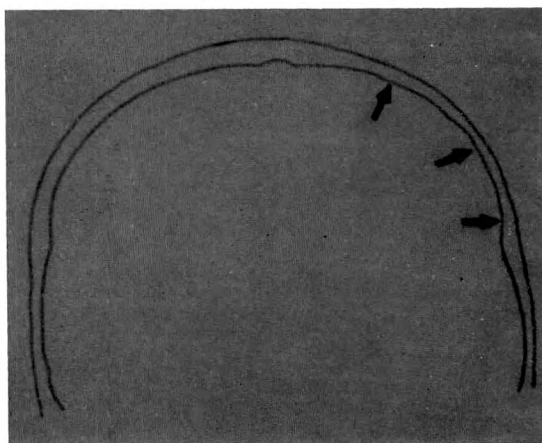


FIG. 16. Thinning of the cranial vault over a superficial malformation (arrows). Tracing of original roentgenogram.



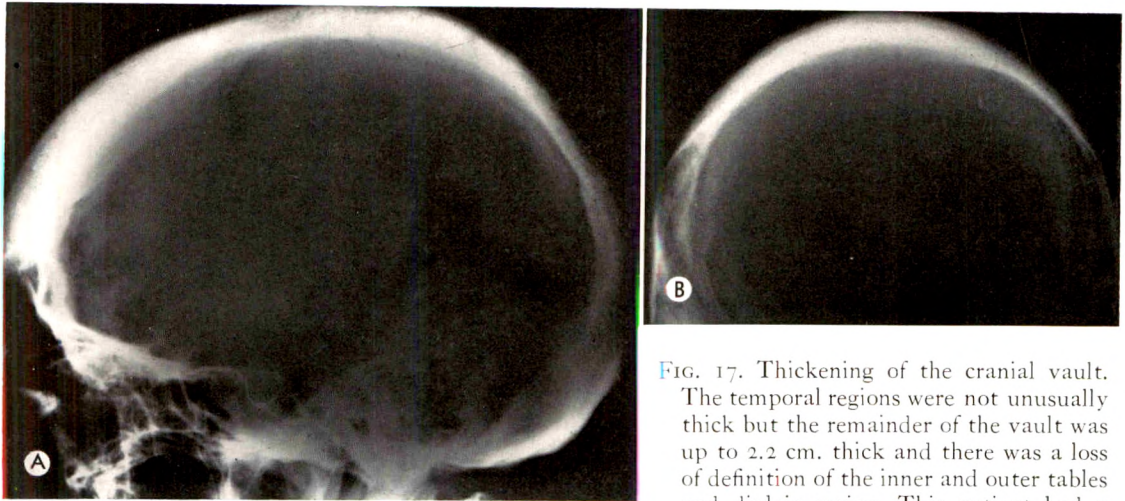


FIG. 17. Thickening of the cranial vault. The temporal regions were not unusually thick but the remainder of the vault was up to 2.2 cm. thick and there was a loss of definition of the inner and outer tables and diploic region. This patient had a

very large malformation supplied by the middle cerebral artery. (A) Lateral view. (B) Frontal view.

tients, but the frequency and prominence of this change in the present series suggest that generalized thickening of the cranial vault may be related to large intracranial vascular anomalies. Well-marked thickening of the cranial vault was also noted in at least 1 of the cases of Olivecrona and Ladenheim.<sup>7</sup> These authors considered that this skull thickening was seen only where the external carotid artery supplied the malformation, but in the present series skull thickening was seen when the malformation was supplied entirely by the internal carotid and vertebral arteries.

Szilagyi *et al.*<sup>11</sup> have reported a 27 per cent incidence of increased length of the lower limb in association with congenital arteriovenous fistulas of the limb. It has been shown by Myers and Janes<sup>6</sup> that the increase of limb length occurs in the tibia and fibula and the work of Weinman *et al.*<sup>12</sup> suggests that the enlargement may be related to alterations of the blood flow, but there was no consistent relationship between change in bone plasma flow and change in bone weight. Keck and Kelly<sup>4</sup> found no increase in bone length in dogs when the venous pressure in a hind limb was increased experimentally. It seems possible that the mechanism of skull thickening in association with large intracranial vascular malformations may be similar to

that involved in these cases of limb hypertrophy.

A second possibility is that a very large vascular malformation may result in a brain of small size and the thick skull is secondary to this change in the brain.

#### DISCUSSION

Small arteriovenous malformations of the brain not associated with an intracranial hematoma rarely show any abnormality in roentgenograms of the skull. Calcification is uncommon in the smaller malformations. Larger malformations are associated with marked enlargement of feeding arteries, draining veins, and venous sinuses, and these, in turn, may cause recognizable changes in the skull. Additional views and sometimes tomography may be required to demonstrate enlargement of the internal carotid artery or jugular veins.

A base roentgenogram or a base tomogram may show enlargement of the vertical portion of the carotid canal within the petrous temporal bone. The portion of the carotid canal passing forward and medially in the petrous temporal bone may sometimes be shown end-on by a lateral view with the face turned 30° away from the side of interest. Due to the variation in this part of the course of the artery, the appropriate position for an axial view of the canal is



variable and some form of fluoroscopic positioning might be helpful. Base roentgenogram or bone tomograms may show an erosion of the lateral aspect of the body of the sphenoid, and frontal tomograms of the sella turcica may show deepening of the carotid grooves. Enlargement of the vertebral arteries causes enlarged foramina in the first cervical vertebra, best shown in a base view or by tomography of the first cervical vertebra in the same position as for a base view of the skull. Enlarged arteries or veins may cause prominent grooves in the cranial vault. These are usually seen in the standard skull roentgenograms. The grooves caused by enlarged venous sinuses are seen in standard skull roentgenograms, but enlargement of the jugular fossae may not be recognized unless a subaxial projection is used, possibly supplemented by tomography.

No normal measurements of the carotid canal have been established and we have had to rely on a subjective impression of enlargement or a gross asymmetry of the right and left internal carotid grooves or canals.

#### SUMMARY

The roentgenographic changes in the skull seen in 61 patients with intracranial arteriovenous malformations are described. Gross enlargement of the internal carotid artery, venous sinuses and veins caused recognizable changes in roentgenograms of the skull. Thickening of the skull was sometimes associated with intracranial arteriovenous malformations. In some cases this involved the hemisphericum on the side of the malformation and in others the cranial vault was thickened on both sides.

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## DYNAMIC DISPLACEMENTS OF INTRACRANIAL STRUCTURES SIMULATING MASS LESIONS

By MANNIE M. SCHECHTER, M.D.,\*† LAWRENCE H. ZINGESSER, M.D.,\*  
and C. CUEVAS, M.D.†  
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ENCASED in bone, tethered by its membranes, and immersed in fluid, the brain is well protected by nature against the ordinary hazards of life. Little movement is allowed the brain providing that the fortress and its contents are intact. However, if the integrity of the bony vault is lost, membranes and brain may herniate through the bony defect. Similarly, tears in the membranes covering the brain, loss of fluid surrounding the organ, and loss of brain substance itself allow varying degrees of freedom of movement.

The freedom of movement allowed the brain when its support is removed facilitates the neurosurgical approach to certain areas of the brain. Positioning of the head allows gravity to assist in retracting a lobe of brain to expose the operative field to better advantage. The use of urea has enhanced these effects. Further extreme freedom of movement is observed by the neurosurgeon who operates on a hydrocephalic and watches the brain collapse like an exhausted balloon when the cerebrospinal fluid drains out of the ventricular system.

It is the purpose of the authors to report certain changes in the roentgenologic ventricular and subarachnoid anatomy of atrophic brains related to the position of the head. These changes were of a magnitude sufficient to cause confusion in diagnosis.

Three cases are reported, one of which showed an aqueduct which assumed an abnormal shape when the patient was placed in the prone position. When the position of the patient's head was changed so that gravity influenced the pons to move in a dorsal direction, the abnormality was no

longer obvious. The second case showed a depression and contralateral displacement of the body of a lateral ventricle during encephalography. Ventriculography, a few days later, demonstrated a normal ventricular system. The third case showed a displaced aqueduct, presumably from the introduction of subdural, subtentorial air, which displaced it downwards and forwards.

### REPORT OF CASES

**CASE 1.** S.H.M. This 43 year old female was admitted to hospital with a history of frontal headaches for 1 year. Four months prior to admission, her symptoms became more severe and she developed difficulty in maintaining her balance as well as dizziness and blurred vision. The cerebrospinal fluid examination was unremarkable. The only positive neurologic finding was an intention tremor of the right hand. The electroencephalogram was normal and the radioisotope scan was unremarkable.

The early pneumoencephalograms showed filling of a large cisterna magna. The fourth ventricle, aqueduct and the back end of the third ventricle appeared normal. A considerable amount of air entered the subdural and subarachnoid spaces and passed over the cerebral cortex (Fig. 1, A-C). Pneumoencephalograms with the patient supine showed the anterior margin of the brain separated by a large margin from the inner table of the skull (Fig. 3, A and B). Air also outlined a large pontine cistern. Roentgenograms taken with the patient prone showed considerable narrowing of the pontine cistern (Fig. 3, C and D).

An autotomogram taken during ventriculography (2 hours later) with the head brow-down showed large lateral ventricles, a large cisterna magna, and outlined the third ventricle, aqueduct and fourth ventricle. The configuration of the aqueduct had altered considerably in this study (Fig. 1, D-F). The basal angle (Sutton) had changed, and was considerably less than

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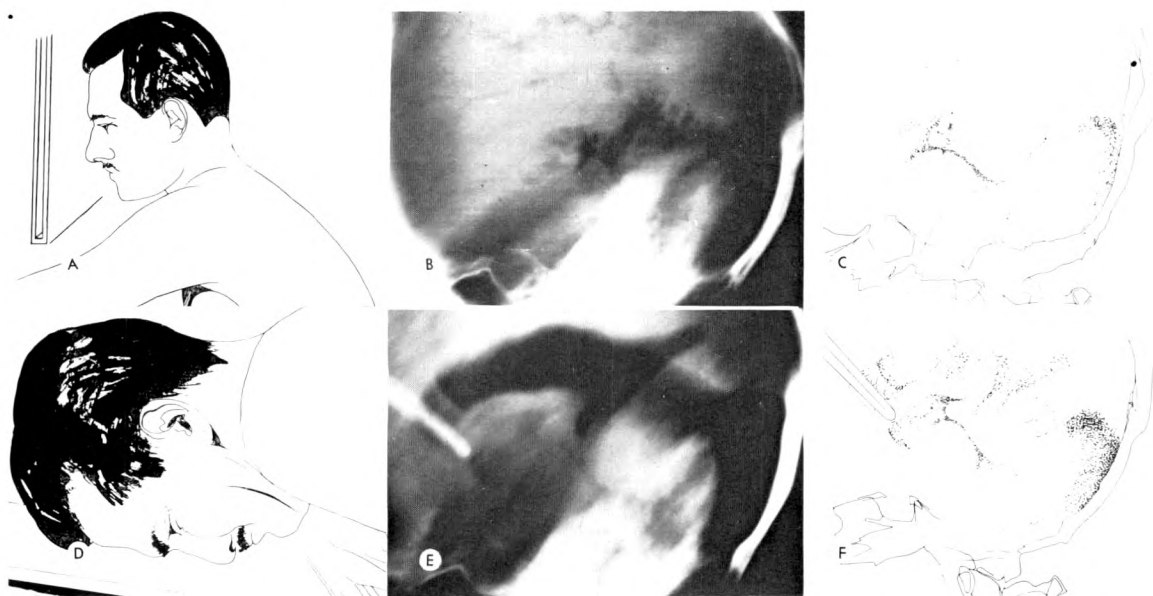


FIG. 1. Case 1. (A-C) Encephalogram—sitting position. Part of the fourth ventricle, the aqueduct and the back end of the third ventricle are outlined. The configuration of the aqueduct appears normal. A capacious cisterna magna is outlined with air. (D-F) Ventriculogram 2 hours later with the patient in brow-down position (autotomogram). Note large lateral ventricles and cisterna magna. The third ventricle, aqueduct and part of the fourth ventricle are outlined. Note that the configuration of the aqueduct has altered and that the ventricular angle has changed significantly. The basal angle (Sutton<sup>7</sup>) is less than  $85^{\circ}$ .

$85^{\circ}$ . In the anteroposterior projection, marked separation of the cortex from the inner table of the skull was seen.

Pantopaque ventriculograms in the supine (Fig. 2, A and B) and prone position revealed similar findings.

Further examinations over a period of time have shown no neurologic abnormality and the patient has remained asymptomatic for a year.

**CASE II.** S.V.H. #201937. This 55 year old female was admitted to hospital 6 months after the onset of progressive deterioration of memory and powers of concentration. The patient also complained of headaches, lethargy and irritability. She frequently became depressed and apathetic. Except for changes in her mental status, the neurologic examination was normal. An electroencephalogram was normal. Plain roentgenograms of the skull were normal and a radioisotope scan was unrevealing. An examination of the cerebrospinal fluid was unremarkable.

Pneumoencephalography showed a remarkable degree of cortical atrophy. Air outlined large dilated sulci, particularly prominent in the parietal region. The basal cisterns were remarkably capacious and air moved freely from front

to back with the patient in the supine and prone positions, respectively (Fig. 4, A and B). The lateral ventricles were dilated and the left lateral ventricle (in its posterior aspect) was shifted to the right of the midline and its roof was depressed. These findings suggested the presence of a mass lesion in the left posterior parietal region (Fig. 4, B and C).

The patient returned to the radiology department 24 hours later, and brow-down studies in the lateral projection revealed that a good deal of the subarachnoid air had been absorbed and the posterior aspects of the left lateral ventricle had assumed a normal appearance (Fig. 5).

Ventriculography performed 48 hours later revealed ventricles which were dilated but normal in position. The shift of the ventricular system to the right of the midline and the depression in the roof of the left lateral ventricle seen in the previous study were no longer present (Fig. 6, A, B and C).

The patient was discharged and a 6 month follow-up revealed no change.

**CASE III.** E.G.B. #17414, G.N.H.C.H. A 67 year old, white male alcoholic was admitted to hospital with 2 months of blurring of vision, and intermittent dizziness.

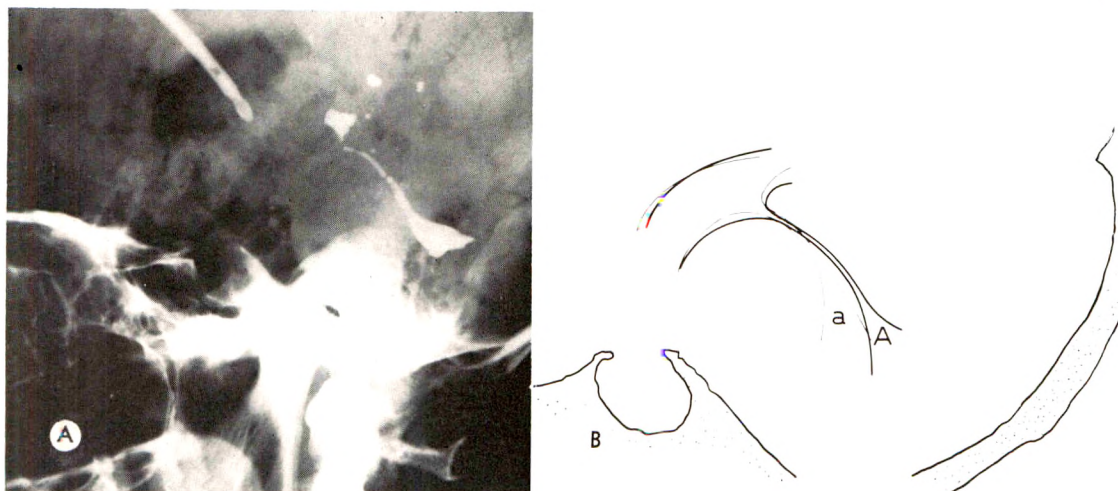


FIG. 2. Case 1. (A and B) Pantopaque ventriculogram with patient in supine position. The aqueduct appears to have a normal course (A). When patient was placed in prone position, the aqueduct changed to (a). Compare with Figure 1, D-F.

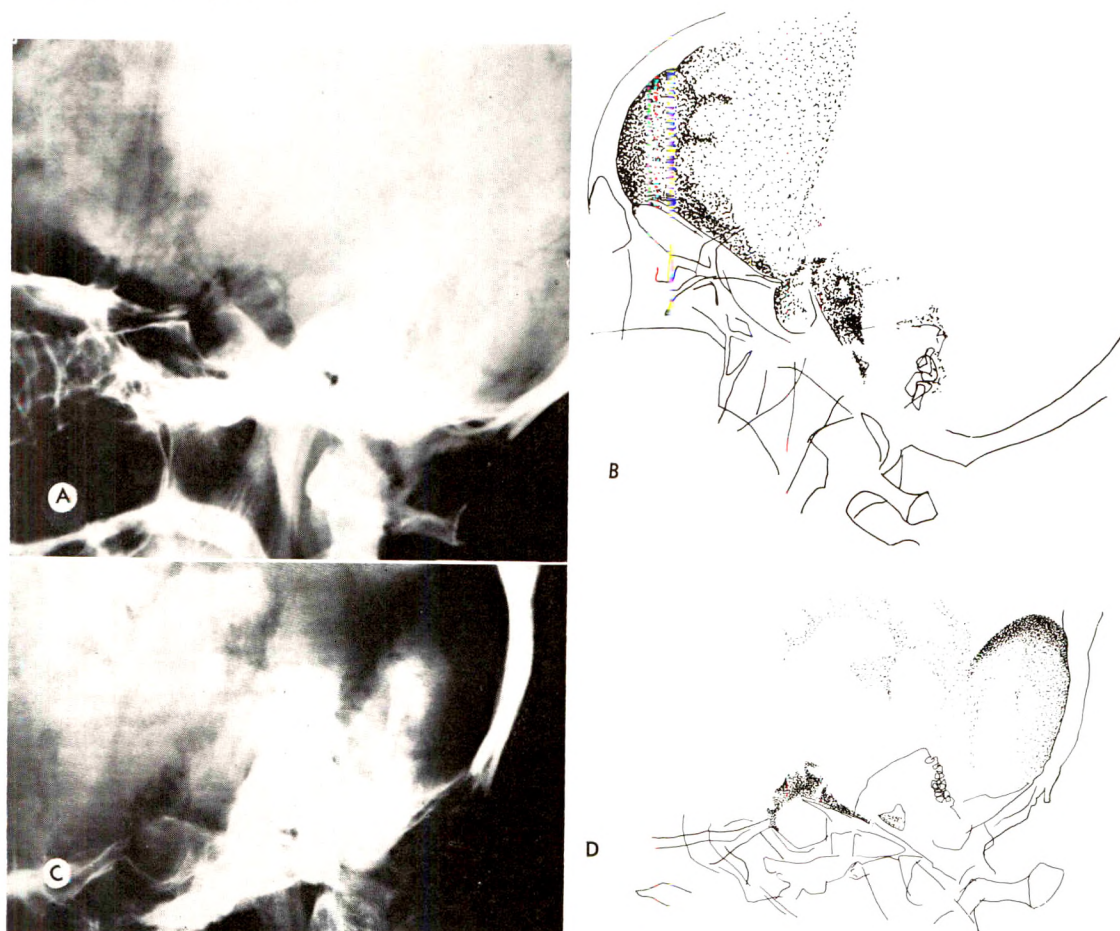


FIG. 3. Case 1. (A and B) Pneumoencephalogram—patient supine. Note that the subarachnoid space in the frontal region is outlined, separating a frontal lobe from the floor of the frontal fossa. Note also the large interpeduncular and pontine cisterns. (C and D) Pneumoencephalogram—patient is prone. Note that the pontine cistern is considerably narrowed as compared with the supine study.



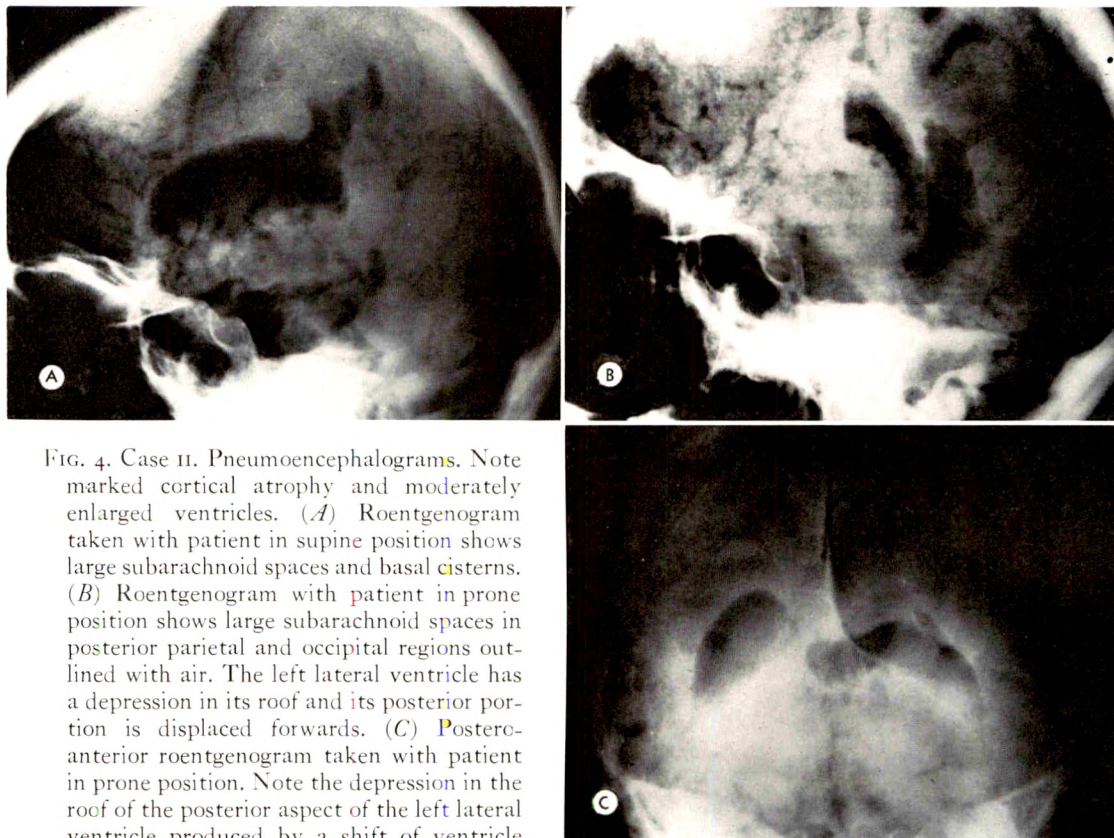


FIG. 4. Case II. Pneumoencephalograms. Note marked cortical atrophy and moderately enlarged ventricles. (A) Roentgenogram taken with patient in supine position shows large subarachnoid spaces and basal cisterns. (B) Roentgenogram with patient in prone position shows large subarachnoid spaces in posterior parietal and occipital regions outlined with air. The left lateral ventricle has a depression in its roof and its posterior portion is displaced forwards. (C) Postero-anterior roentgenogram taken with patient in prone position. Note the depression in the roof of the posterior aspect of the left lateral ventricle produced by a shift of ventricle to the right under the falx.

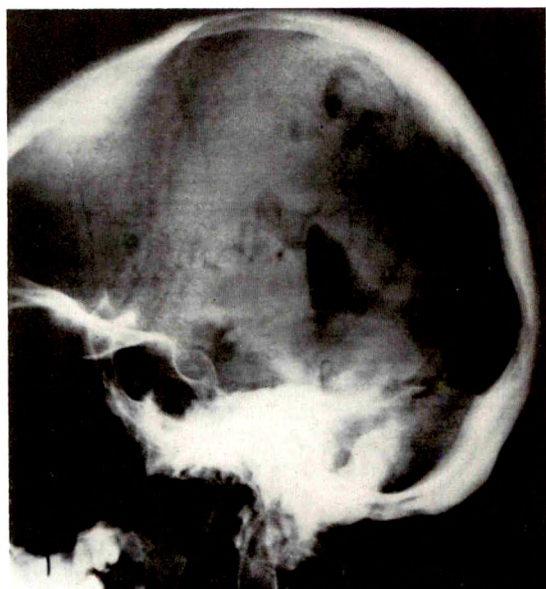


FIG. 5. Case II. Prone roentgenogram made 24 hours after Figure 4, A, B and C. The posterior aspect of the left lateral ventricle has assumed a more normal position.

During encephalography, air passed via the basal cisterns to the fourth ventricle, aqueduct, third ventricle and lateral ventricles. A considerable amount of air passed into the subdural space and collected in the subtentorial region. At the level of the inferior colliculus, the aqueduct was kinked. This kink simulated the type of angulation seen with a cerebellar mass (Fig. 7).

#### DISCUSSION

The 3 cases reported here demonstrate that movements of the cranial contents are influenced by gravity. These movements are the result of several factors which include disproportion between the size of the cranial cavity and its contents and air in the extracerebral spaces (instead of the cerebrospinal fluid which acts as a cushion). An appreciation of shifts of the intracranial structures in the absence of mass lesions obviated serious misinterpretations.

In 1960 Breig<sup>1</sup> documented movements



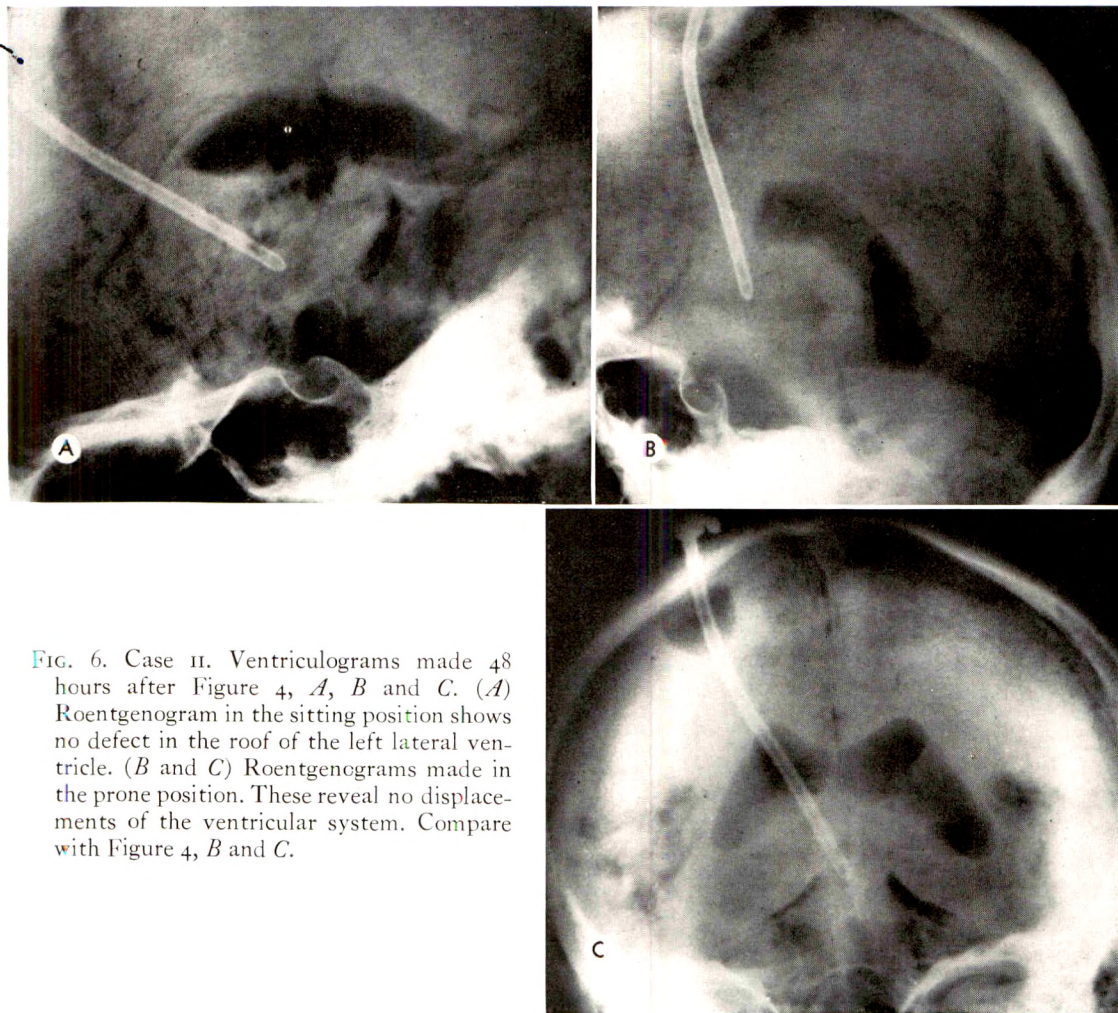


FIG. 6. Case II. Ventriculograms made 48 hours after Figure 4, A, B and C. (A) Roentgenogram in the sitting position shows no defect in the roof of the left lateral ventricle. (B and C) Roentgenograms made in the prone position. These reveal no displacements of the ventricular system. Compare with Figure 4, B and C.

and distortions of the normal hind brain with flexion and extension of the head at the atlanto-occipital junction. Observations were made on living subjects and cadavers. In living subjects, roentgenograms were taken during encephalography with the head in extension and flexion. Observations were also made during surgery where the anatomy of the posterior fossa structures was observed during extension and flexion of the neck. Cadaver studies included roentgenograms taken of anatomic landmarks identified by radiopaque markers. The anatomy was also exposed *in situ* and the effects on movements were documented. Deformations and changes in the position

of the fourth ventricle were noted in extension and flexion.

Diekman, Schmidt and Prager,<sup>3</sup> in an analysis of 1,800 stereotaxic brain operations performed after pneumoencephalography, found that air had collected in the extracerebral space in 13 per cent of cases before the skull was opened. When extracerebral air more than 4 mm. in width was present, gravity influenced changes of intracranial structures. The position of the calcified pineal gland relative to fixed bone landmarks was compared in the sitting position and with the patient supine. Significant changes in the position of the pineal gland occurred.





FIG. 7. Case III. Pneumoencephalogram. Air has entered the fourth ventricle, aqueduct, and back end of the third ventricle and lateral ventricles. A considerable amount of air has passed under the tentorium. Note the kink in the proximal course of the aqueduct, produced by the presence of sub-tentorial air.

Liliequist,<sup>5</sup> in 1959, found a significant difference in the size of the pontine cistern with the patient in the sitting position and with the patient in the supine position. He showed that when the patient sits with his head flexed, the brain sinks slightly, causing flattening of the pontine cistern. In 1897 Forriep<sup>4</sup> reported that shifts of the intracerebral structures occur with loss of fluid in the postmortem state.

In 1962 Van Buren and MacCubbin<sup>10</sup> compared the positions of intracerebral structures outlined during encephalography after taking roentgenograms with the patient's head in the brow-up and brow-down position. Although their findings suggested that no significant movement of the brain occurs with position during pneumoencephalography, they state that the introduction of subdural air, as often occurs in ventriculography, may result in a significant shift of intracerebral structures.

The 3 cases here reported all showed a remarkable degree of cerebral atrophy as outlined by the air studies. In Case I with the patient lying supine, the marked disproportion between the volume of the cerebral contents and the cranial cavity al-

lowed the frontal lobes to fall away from the frontal bone. Note that a capacious pontine cistern was present. With the patient sitting erect and with the chin tucked in, the frontal lobes dropped forward and obliterated the large frontal subarachnoid spaces. Note that the pontine cistern also was narrow. When the patient was placed brow-down, the cerebellum moved forward and the ventricular angle (Sutton<sup>7</sup>) became abnormally small. The ventricular angle is an angle between a line joining the mid-point of the floor of the fourth ventricle to the mouth of the aqueduct and another from here to the lowest point of the floor of the third ventricle. This angle (in the normal encephalogram) is remarkably constant at about 90°. The range of variation is between 85° and 95°. The course and shape of the aqueduct, a sensitive and reliable index of space taking lesions in the region of the tentorial opening and in the posterior fossa,<sup>2,6,8,9</sup> must be carefully evaluated in the light of the above observations.

In Case II again there was a considerable disproportion between the volume of cerebral tissue and the cranial vault. In the brow-down position, part of the left cerebral hemisphere passed under the falx into the right side. This produced a depression in the roof of the posterior portion of the left lateral ventricle.

A roentgenogram taken 24 hours later, when a considerable amount of extraventricular air had been absorbed, appeared normal. Furthermore, ventriculography was performed 48 hours later and the findings were no longer equivocal. The back ends of the lateral ventricles were undisplaced.

Case III resembles the case report of Liliequist.<sup>5</sup> Here an accumulation of sub-tentorial air had altered the configuration of the aqueduct.

#### SUMMARY

Changes occur in the roentgenologic anatomy of the ventricular system and subarachnoid spaces, depending upon the position of the head. These changes, influenced by gravity, are most marked when

there is a significant disproportion between the volume of the cranial vault and the cerebral tissue, and may simulate mass lesions.

Three cases are reported where these changes were of sufficient magnitude to cause confusion in diagnosis.

The pertinent literature on the subject is reviewed.

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We are grateful to Doctor James Scatliff for Case III.

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## THE DIFFERENTIATION OF EXTRINSIC FROM INTRINSIC INTRACRANIAL TUMORS WITH PARTICULAR REFERENCE TO POSTERIOR FOSSA TUMORS

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ONE of the more important aspects in the investigation of intracranial tumors is to distinguish extrinsic masses which are almost always benign from intrinsic tumors which are almost always malignant. The same consideration applies to intraventricular tumors, some of which also are benign. This distinction can almost always be made, but may require the use of several diagnostic techniques.

Ventriculography, since it fails to distinguish extrinsic from intrinsic tumors, is not the most informative procedure, but may complement other findings. Equally, ventriculography may fail to differentiate between extrinsic and intraventricular tumors.<sup>7</sup> Encephalography, on the other hand, provides a most reliable criterion of extrinsic origin by outlining a mass within the subarachnoid space. Basal and posterior fossa tumors, in particular, lend themselves to identification in this way. Angiography may supplement these findings by revealing the nature of the mass (Fig. 1, *A*, *B* and *C*).

Selective angiography of the external carotid artery also permits differentiation of extrinsic from intrinsic tumors, and is of value in supratentorial as well as infratentorial masses (Fig. 2).<sup>6</sup> Failure to demonstrate the external carotid circulation may result in a tumor being thought malignant when it is benign, and *vice versa*. Negative findings may be of value.

### POSTERIOR FOSSA TUMORS

The posterior fossa is small with well defined and constant landmarks provided by the subarachnoid cisterns. For this reason encephalography is the most logical procedure in the investigation of posterior fossa

tumors.<sup>4,5</sup> It is of the utmost importance to distinguish extra-axial masses from intrinsic tumors. The former may be outlined within the basal cisterns. Whereas extra-axial masses tend to enlarge the cistern in which they are situated, intra-axial masses tend to compress it. Dilatation of the subarachnoid cisterns may be found supratentorially,<sup>9</sup> or on the side opposite an intra-axial mass.

The posterior fossa encephalogram is essentially a preoperative procedure, and cannot be performed unless neurosurgical facilities are available. In some cases, it has been our practice to place preliminary burr holes, or alternatively, twist drill burr holes, enabling one to tap the ventricles should the occasion arise. Air is injected extremely slowly in 7 cc. increments without withdrawal of cerebrospinal fluid. Each stage is monitored with posteroanterior and lateral roentgenograms, autotomography being an integral part of the procedure.

The cerebellar tonsils are readily identified on the autotomogram<sup>11</sup> (Fig. 3). If herniated, this is evidence of a posterior fossa tumor, although occasionally a supratentorial mass may cause tonsillar herniation. An Arnold-Chiari malformation must be differentiated.<sup>3</sup> An inferior fourth ventricle tumor presenting in the upper cervical canal can be distinguished from the cerebellar tonsils and the cerebellum.<sup>10</sup> The vallecula, if filled, is displaced by cerebellar masses localizing their site, or dilated by a protruding fourth ventricle tumor.

The cerebellopontine angle cisterns must be demonstrated. A normal cerebellopontine angle cistern excludes a cerebellopontine angle mass. The same consideration

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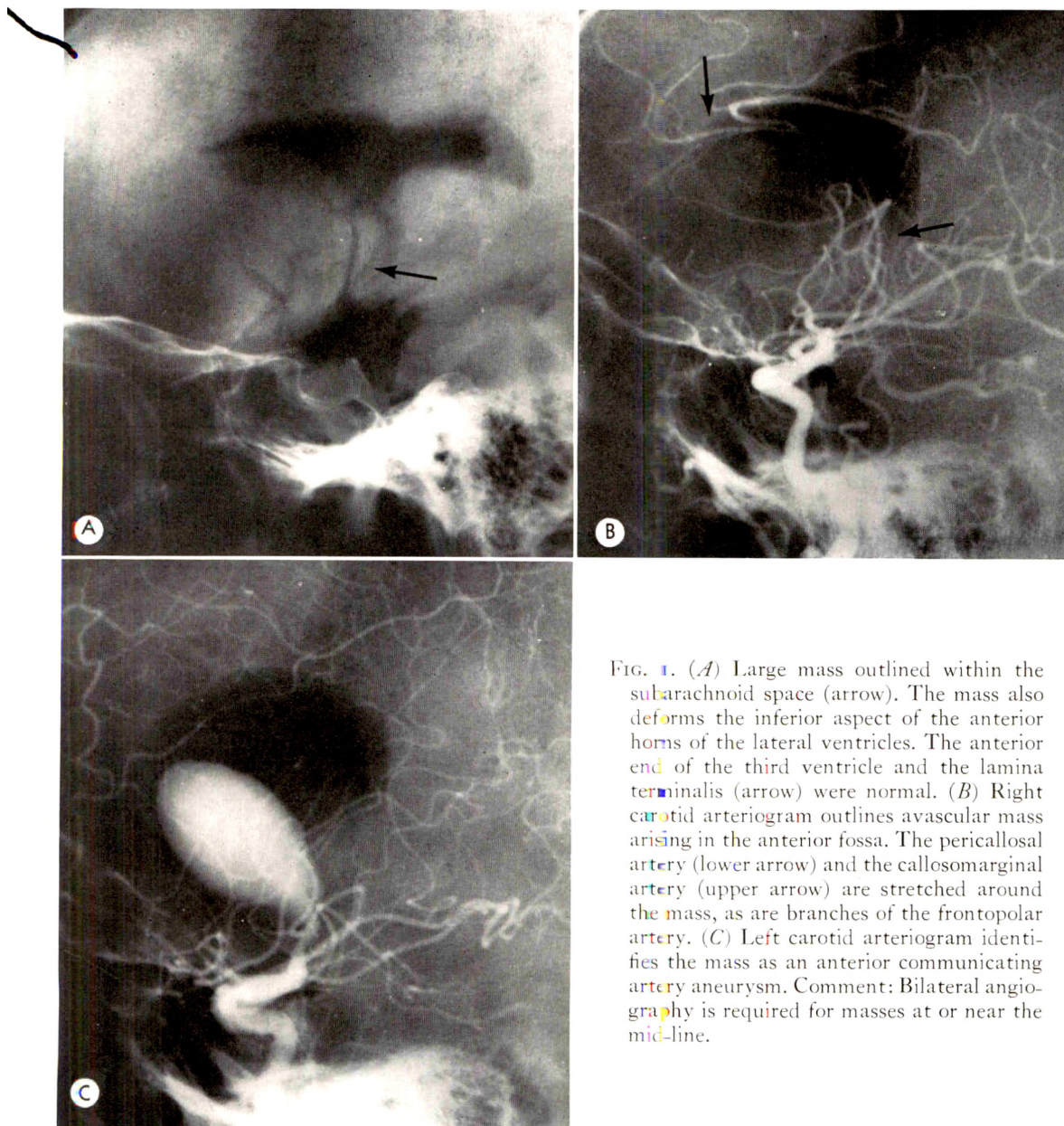


FIG. 1. (A) Large mass outlined within the subarachnoid space (arrow). The mass also deforms the inferior aspect of the anterior horns of the lateral ventricles. The anterior end of the third ventricle and the lamina terminalis (arrow) were normal. (B) Right carotid arteriogram outlines avascular mass arising in the anterior fossa. The pericallosal artery (lower arrow) and the callosomarginal artery (upper arrow) are stretched around the mass, as are branches of the frontopolar artery. (C) Left carotid arteriogram identifies the mass as an anterior communicating artery aneurysm. Comment: Bilateral angiography is required for masses at or near the mid-line.

applies to the pontine cistern. Intrinsic tumors of the pons, cerebellum, or posteriorly situated masses compress the pontine angle cisterns. Cerebellar tumors may also compress the ipsilateral ambient cistern. Failure to demonstrate these cisterns requires a more determined attempt to fill them. An extrinsic mass lesion can only be diagnosed if one has been outlined within a cistern (Fig. 4, A and B; 5; and 6).

Cerebellopontine, prepontine, pontine and even suprasellar masses may all deform the aqueduct in the same way by showing a minimal increase in its normal curvature, so that ventriculography is not helpful in determining their origin (Fig. 4, A and B; and 7, A, B and C). Cerebellopontine masses may not deform the ventricular system at all, so that ventriculography is potentially misleading. While the majority of



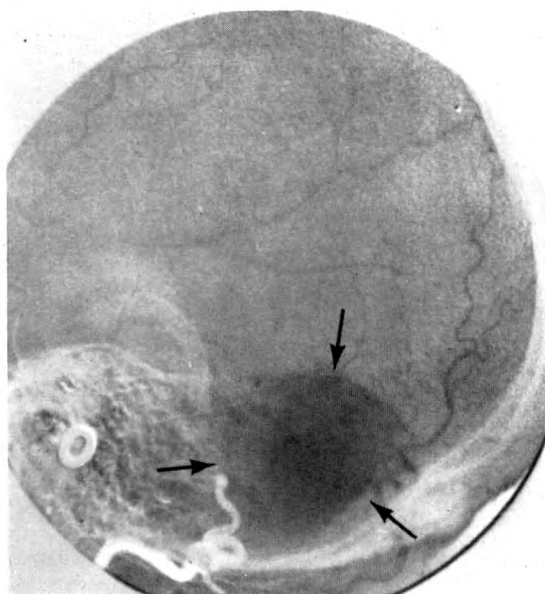


FIG. 2. Selective external carotid arteriogram demonstrating posterior fossa meningioma (arrows) which derives its blood supply from the occipital artery.



FIG. 3. Autotomogram to show cerebellar tonsil at its junction with the cerebellar hemisphere (arrow) during initial stage of air encephalography.

acoustic neuromas show bone changes, these tumors may be bilateral, may cause no bone erosion, may cause no ventricular displacement, or may show changes which, at ventriculography, are indistinguishable from those produced by pontine tumors. On the other hand, they may be outlined within the cerebellopontine angle cistern at

encephalography; bilateral lesions may be demonstrated in the same way (Fig. 4, *A* and *B*; 5; and 6).

Positive contrast encephalography<sup>8</sup> is a very elegant way of demonstrating extremely small angle tumors. However, since the procedure anticipates the diagnosis and does little to exclude other possibilities, it is recommended only as a complementary investigation when air studies have failed.

Prepontine lesions, such as chordoma, meningioma, or basilar aneurysm, may be

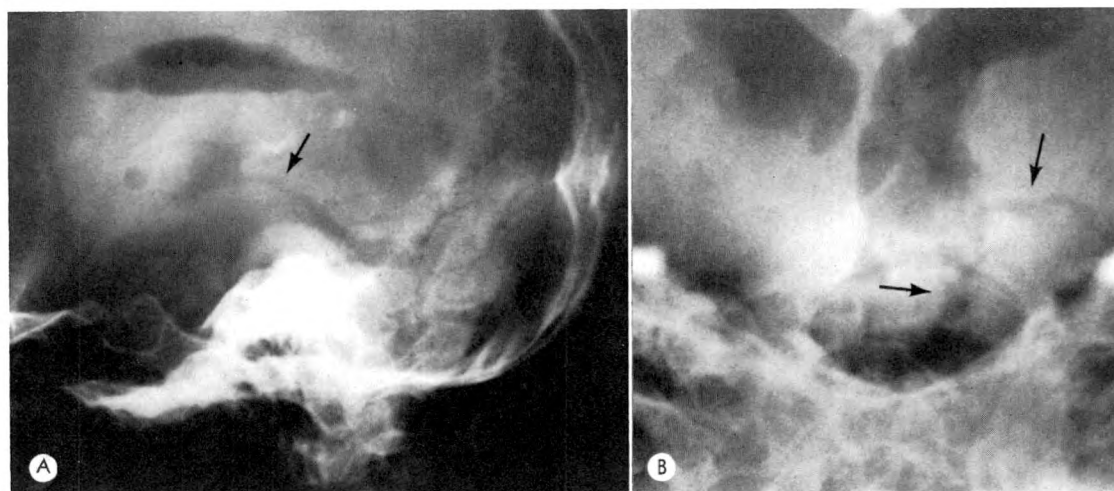


FIG. 4. Acoustic neuroma. (*A*) Air encephalogram during the filling stage shows the increased curvature of the aqueduct (arrow). (*B*) Acoustic neuroma outlined within the cerebellopontine angle cistern (arrows).

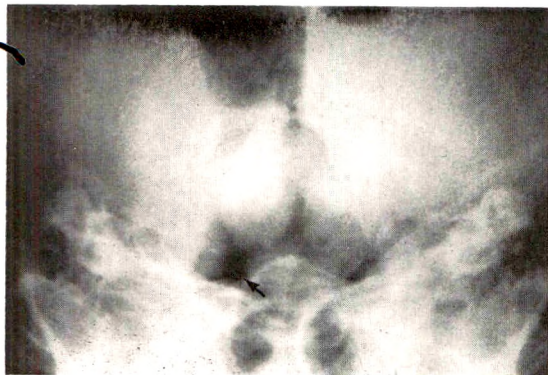


FIG. 5. Acoustic neuroma outlined within the cerebellopontine angle cistern (arrow).

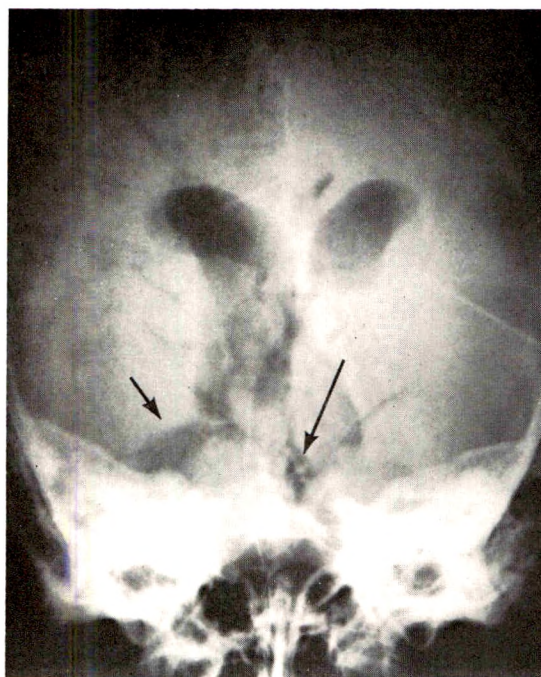


FIG. 6. Chordoma. The mass fills both cerebellopontine angle cisterns. Left short arrow shows dilated cerebellopontine angle cistern beyond the mass. Long right arrow demonstrates the basilar artery, and just lateral is the medial margin of the mass in the right cerebellopontine angle cistern.

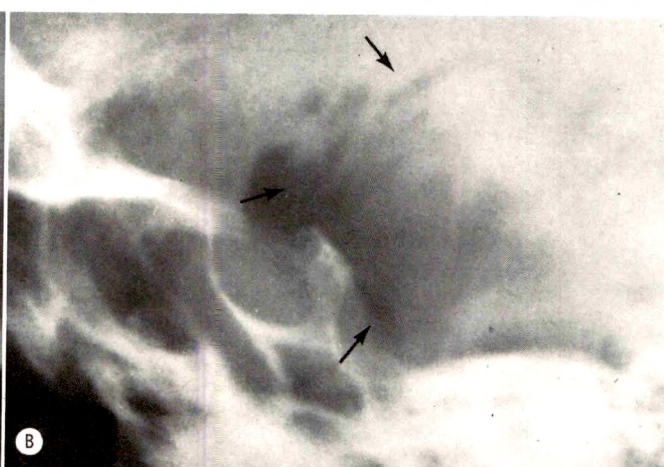
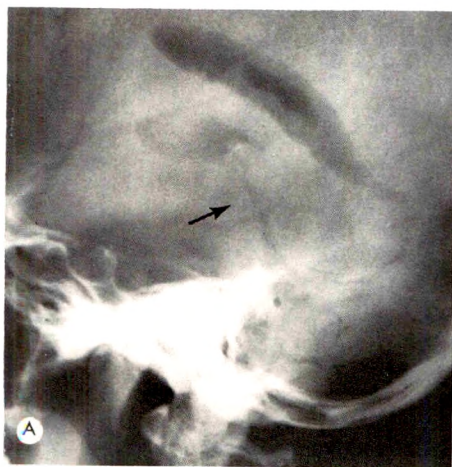
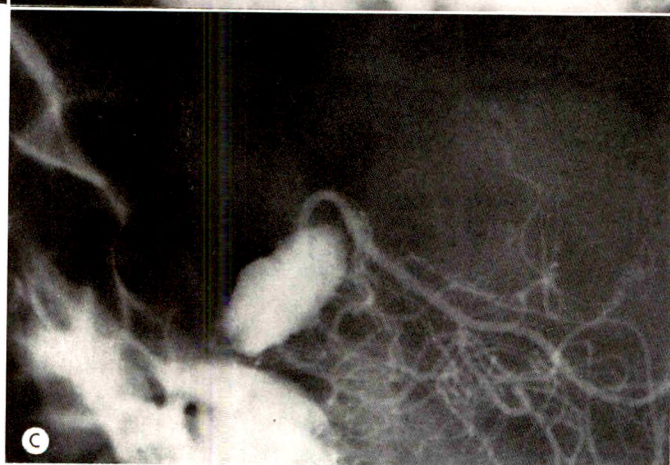


FIG. 7. Basilar artery aneurysm. Female, aged 34 years. (A) Air encephalogram during filling stage shows the slight backward displacement of the aqueduct (arrow). (B) Mass outlined within the pontine cistern (upper and lower arrows). The middle arrow shows the mammillary bodies just above it and the third nerve just in front of the mass and above the posterior clinoid processes. (C) Vertebral arteriogram shows basilar artery aneurysm. Comment: Vertebral angiography is required for any possibly vascular extrinsic mass in the pontine or cerebellopontine angle cisterns.





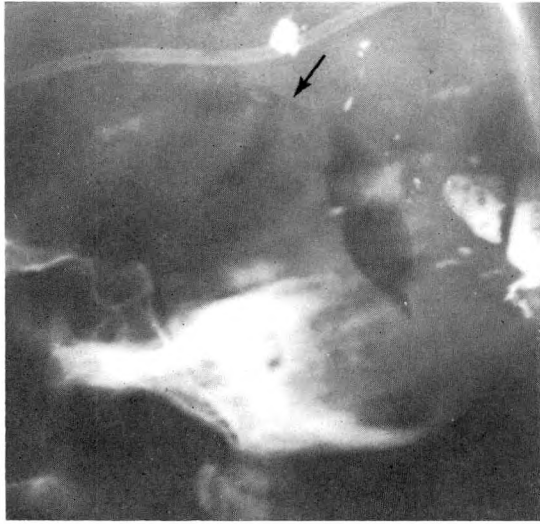


FIG. 8. Air encephalogram in a case of pontine tumor. The pontine cistern is very shallow. The quadrigeminal plate cistern shows upward and backward displacement (arrow). The fourth ventricle is dilated, stretched around the pontine mass and displaced backwards.

outlined within the pontine cistern and shown to be extra-axial (Fig. 7, *A*, *B* and *C*). Chordomas may show neither calcification nor destructive changes in the early stages, although later both may be present

(Fig. 6). Pontine tumors seldom give rise to difficulty since ventricular filling is obtained even when these tumors are very large (Fig. 8). Pontine tumors displace the aqueduct and fourth ventricle posteriorly, and the diagnosis can be made when the changes are quite subtle, at a time when the usual measurements are still within normal limits. As the tumor enlarges, the floor of the third ventricle is elevated and displacement of the aqueduct and fourth ventricle becomes more marked. These changes, too, may be present with extra-axial masses.

The differentiation of pontine and fourth ventricle tumors, at ventriculography, may present difficulty since an incompletely filled fourth ventricle may be mistaken for a displaced fourth ventricle. Complete filling of the fourth ventricle is assured with encephalography. In some cases of fourth ventricle or inferior vermis tumors, no filling of the fourth ventricle is obtained, but the tumor may be outlined in the upper cervical canal.<sup>10</sup> Ventriculography may be required when encephalography is inconclusive.

Displacement of the aqueduct by superior vermis and cerebellar tumors is well

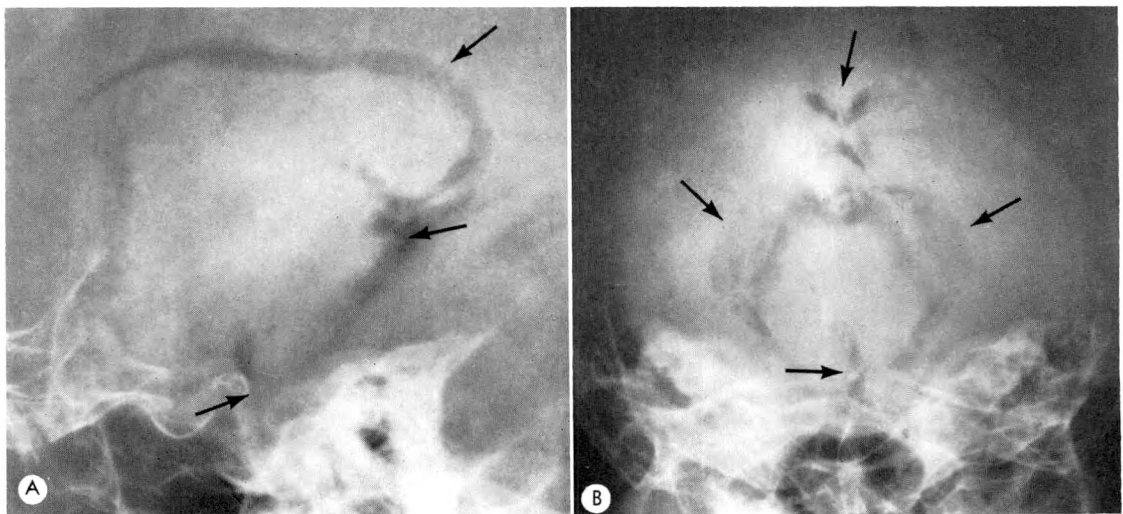


FIG. 9. Right cerebellar tumor. (*A*) There is acute angulation of the quadrigeminal plate between the superior and inferior colliculi (middle arrow). The pontine cistern (lower arrow) is obliterated, and the callosal cistern (upper arrow) is dilated. (*B*) Frontal view shows dilated callosal sulci (upper arrow) and dilated ambient wings (right and left middle arrows). The vallicula shows slight displacement towards the left (lower arrow).

documented, but similar changes in the quadrigeminal plate have received less attention. Cerebellar tumors deform the aqueduct by increasing the normal aqueduct kink between the superior and inferior colliculi. The resultant angulation of the quadrigeminal plate can be demonstrated by air in the quadrigeminal plate and lingular cisterns. In cases where there is no ventricular filling, these findings are diagnostic (Fig. 9, *A* and *B*; and 10).

Cerebellar hemisphere and superior vermis tumors displace the lingular cistern and the inferior colliculi forwards, whereas mid-brain, pontine and prepontine masses displace them backwards. Similar displacement may be observed in inferior vermis and fourth ventricle tumors. In these cases, the lingular cistern is obliterated due to enlargement of the fourth ventricle and an outpouching of the anterior medullary velum. The tumor may also be demonstrated within the cervical subarachnoid space.

Mesencephalic tumors deform the brain

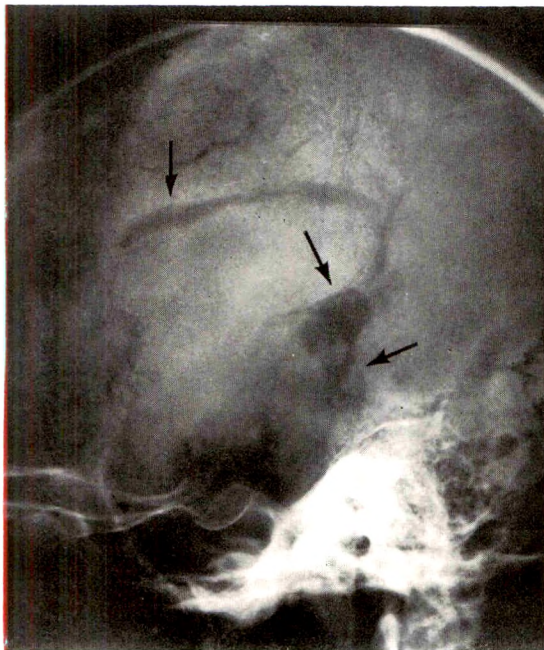


FIG. 10. Gross forward displacement of the quadrigeminal plate by a cerebellar tumor (lower arrow). The callosal cistern is dilated (upper arrow), and also the cisterna venae magnae (middle arrow). The pontine cistern is obliterated.

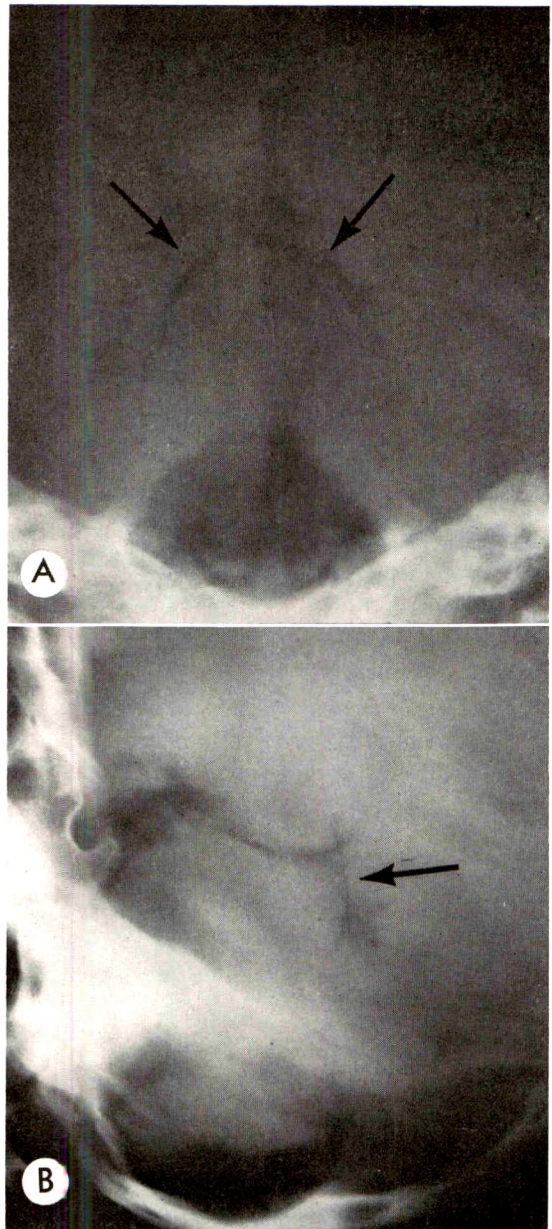


FIG. 11. Mesencephalic tumor. (*A*) Frontal view shows asymmetry of quadrigeminal plate cisterns (arrows). (*B*) Lateral view shows backward displacement of the quadrigeminal plate cistern (arrow) and thickening of the mid brain and pons.

stem and show asymmetry of the quadrigeminal plate and ambient cisterns (Fig. 11, *A* and *B*). Collicular tumors cause aqueduct obstruction and are characterized by thickening of the quadrigeminal plate (Fig. 12). This increases the distance between the



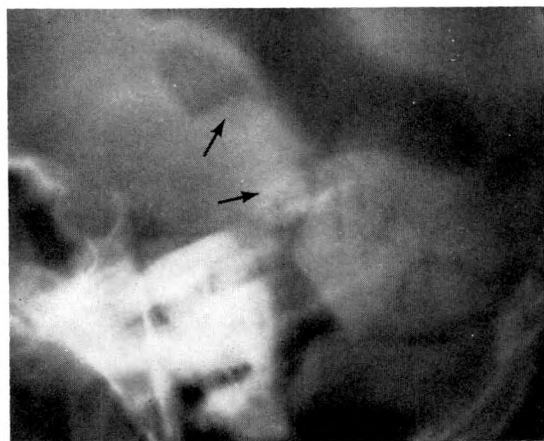


FIG. 12. Lateral filling encephalogram in case of quadrigeminal plate tumor demonstrates the thickening of the quadrigeminal plate (arrows).

aqueduct and the quadrigeminal plate cistern. These tumors must be differentiated from tentorial meningiomas which elongate the aqueduct and displace the fourth ventricle downwards and forwards. Care must be taken not to confuse air outlining a tumor mass with displacement of the quadrigeminal plate cistern.

The role of vertebral angiography is essentially complementary: in prepontine or pontine angle masses, to exclude basilar aneurysms which are being recognized with increasing frequency and which respond poorly to radiotherapy. In obscure fourth ventricle tumors, vertebral angiography

may be helpful in disclosing an unsuspected angioma (Fig. 13, *A* and *B*). Meningiomas and hemangioblastomas may also be elegantly demonstrated by vertebral angiography. Acoustic neuromas may show abnormal vascularity or displacement of the superior cerebellar artery and the petrosal vein.

Selective angiography of the external carotid artery may be helpful in the diagnosis of posterior fossa meningiomas, some of which are nourished by the occipital artery (Fig. 2).

Tentorial meningiomas may also receive blood supply from the tentorial artery,<sup>1</sup> but so may angiomas and malignant tumors invading the dura. At vertebral angiography tentorial meningiomas show separation of the superior cerebellar arteries from the posterior cerebral arteries in the lateral projection.<sup>2</sup> The medial posterior choroidal vessels and the internal cerebral vein may show forward displacement (Fig. 14). Malignant supratentorial tumors extending through the tentorium may also separate the superior cerebellar from the posterior cerebral arteries but, in frontal projection, displace the calcarine branches medially. Separation of the proximal posterior cerebral arteries in the frontal projection is not helpful as this may occur in intrinsic brain stem tumors, but lateral displacement of

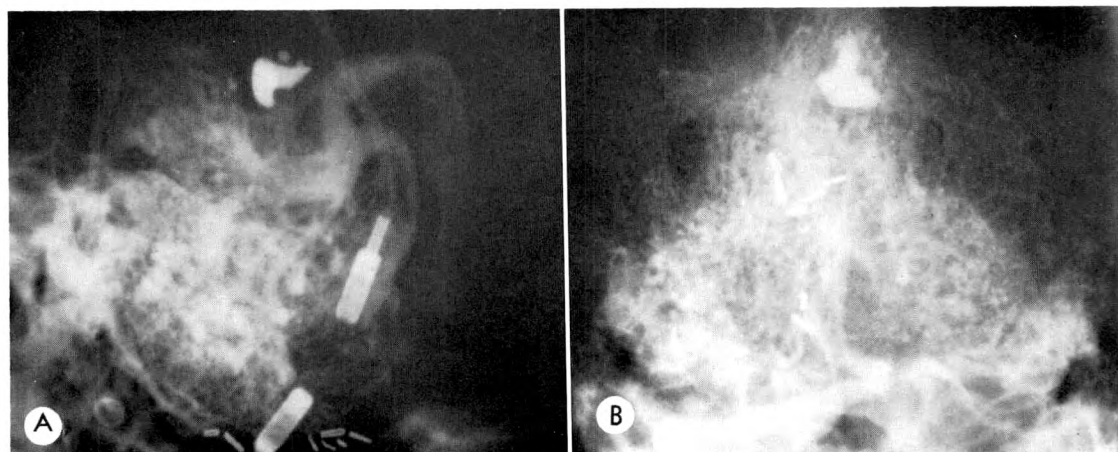


FIG. 13. (*A* and *B*) Vertebral arteriograms demonstrating extensive posterior fossa angioma which involves the fourth ventricle.

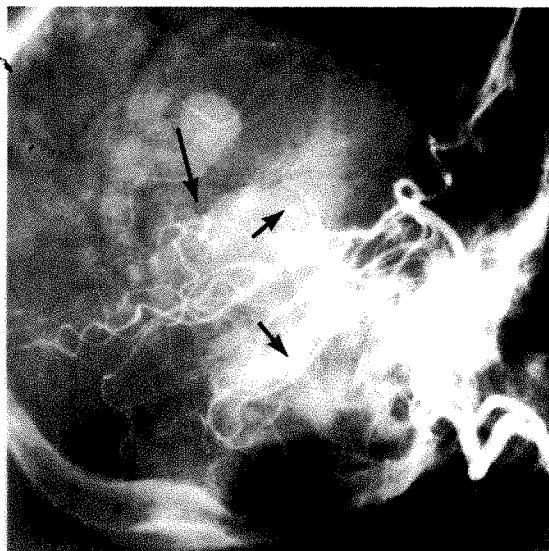


FIG. 14. Tentorial meningioma. Vertebral arteriogram shows cluster of abnormal vessels derived from the posterior cerebral artery and supplying the tumor (upper arrow), forward displacement of the medial choroidal arteries (middle arrow), and downward displacement of the superior cerebellar arteries (lower arrow).

the calcarine branches can only be caused by an extracerebral mass.

#### CONCLUSION

The most logical sequence in the investigation of posterior fossa tumors is air encephalography, possibly supplemented by angiography. At times, it may be necessary to proceed to ventriculography and positive contrast studies.

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## CRANIAL AND INTRACRANIAL LESIONS IN NEUROFIBROMATOSIS\*

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**N**EUROFIBROMATOSIS (von Recklinghausen's disease)<sup>11</sup> is an uncommon congenital and familial disease of tissues of neuro-ectodermal and mesenchymal origin. Classically, the affected patient presents with patches of cutaneous pigmentation (café au lait spots), multiple cutaneous elevated tumors (fibroma molluscum) and neurofibromas of peripheral nerves. Patients not uncommonly show cosmetically deforming and bulky plexiform neuromas, especially about the head and neck or lower extremities.

Roentgenographically, there is often a number of fairly characteristic skeletal deformities. These include spinal scoliosis; bowing, distortion, local giantism and pseudoarthrosis of long bones; elephantoid overgrowth of skin and soft tissues; and local cystic or erosive changes of bone. The roentgenologic aspects of such skeletal lesions have been reviewed in detail by Hunt and Pugh.<sup>5</sup>

The skull may also reflect this condition in a number of ways. Bony cranial marks of the disease, however, are uncommon compared to involvement of the axial and peripheral skeleton. Skull changes may include bizarre deformities of the calvarium and facial bones; erosion and enlargement of skull foramina (especially due to cranial nerve neurofibromas); and ovoid bone defects of the skull vault, especially along the parietal bones and left lambdoid suture. The greater and lesser sphenoid wings are more commonly involved, resulting in changes of orbital walls and sella turcica, sometimes with associated exophthalmos as first described by Le Wald<sup>7</sup> in 1933. Burrows<sup>2</sup> discussed these changes in detail in 1963. Recently, Joffe<sup>6</sup> wrote about patients he observed with a previously un-

documented mark of the disease: cyst-like defects in the lower one-third of the left lambdoid suture. These patients had no overlying cutaneous tumors. Each had other classic marks of the disease, but no mention was made of signs or symptoms of associated intracranial tumors.

The purpose of this presentation is to document and illustrate a number of interesting skull and intracranial lesions of neurofibromatosis as seen in a university teaching center, including the more common acoustic neurinomas and optic gliomas. Various other intracranial tumors have been found in patients with neurofibromatosis, including meningiomas (usually of the posterior fossa and often multiple), and a variety of gliomas, particularly the astrocytomas and ependymomas, as documented by Russell and Rubinstein.<sup>10</sup> Peripheral neurofibromas are frequent in this disease while cord tumors, such as ependymomas and astrocytomas, have been encountered occasionally (rarely associated with syringomyelia).

### ILLUSTRATIVE CASES

**CASE 1.** S.L., Hosp. No. 62-12775. This 12½ year old female presented in 1964 with a left cervical plexiform neuroma, first noted at the age of 6 months. Her mother, 1 brother and 1 sister showed signs of neurofibromatosis. The patient had had three partial removals of the tumor by the time she was 8 years old, including one by intraoral approach. A left radical neck dissection, done at the age of 12½ years, relieved her cosmetic deformity and dysphagia. Skull roentgenograms at 8 years of age (Fig. 1, A and B) showed a well defined bone defect of the left lambdoid suture with left zygomatic and mandibular deformities. The patient died at 13½ years of age in another hospital with symptoms of diplopia and vomiting of short

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duration (? intracranial tumor). An autopsy was not permitted.

CASE II. C.A., Hosp. No. 62-19132. This 34 year old single white female entered the hospital 4 days before death. No familial history of the disease could be obtained. At birth, a neurofibroma was noted in the left side of the patient's tongue. This was later removed. The diagnosis was established at the age of 16 years, with note of cutaneous nodules and café au lait spots. She was mentally retarded. Left temporal hemianopsia was observed at the age of 16 years. Increasing left hemiparesis and facial atrophy were observed by the age of 20 years. During another hospitalization, pneumoencephalography showed evidence of an enlarged right lateral ventricle.

She was admitted because of progressive headache, nervousness, ataxia and dysarthria of several months' duration at the age of 34 years. Skull roentgenograms on the second day of hospitalization (Fig. 2A) demonstrated a left lambdoid bone defect. Mercury 203 isotopic brain scans (Fig. 2, B and C) revealed an increased pattern of activity in the region of the right basal ganglia. Cerebral arteriography suggested lateral displacement of the right lenticulostriate arteries only. Following rapid onset of coma, death occurred.

An autopsy, a Grade I ( $3 \times 3 \times 2$  cm.) fibrillary astrocytoma was found, extending from the right cerebral peduncle into the right globus pallidus, external capsule, thalamus, hypothalamus, and right optic nerve. There was an ovoid defect in the left occipital and parietal bone, bridging the lambdoid suture,  $1 \times 2$  cm. in dimension, with progressive bone thinning toward the center of the defect. The bone was 1 mm. thick at the center of the defect, without under or overlying tumor, cyst, or evidence of meningeal change.

CASE III. J.E.H., Hosp. No. 62-03343. This 7 year old white male entered the hospital with undiagnosed progressive failing vision of both eyes (R>L) for at least 5 years. There were numerous café au lait spots over the body. Skull roentgenograms (Fig. 3A) showed coronal suture diastasis, a "J" shaped sella turcica with depression of the chiasmatic groove and anterior and posterior clinoid erosion. A posterior suprasellar calcification was noted to the left of midline. Views of the optic foramina (not

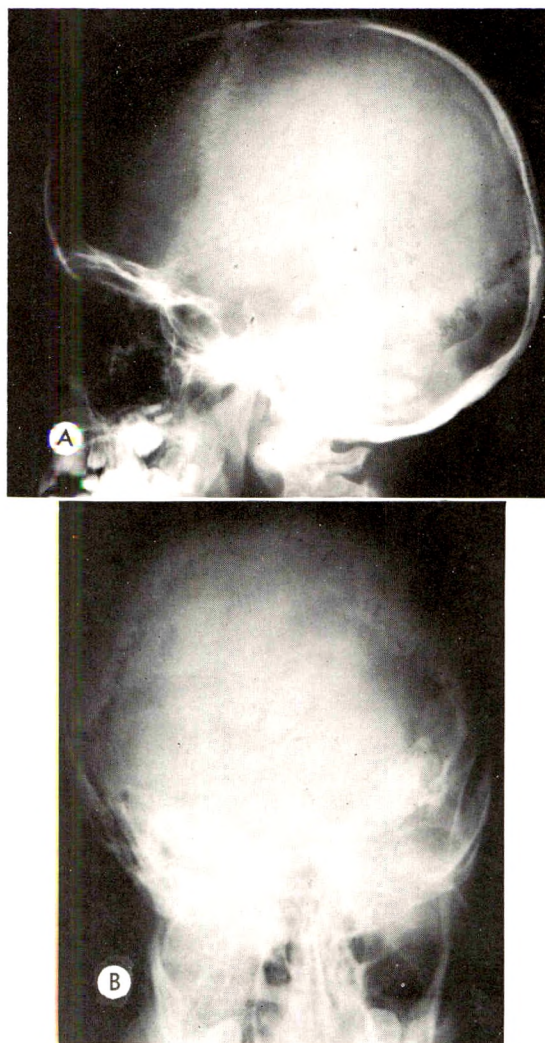


FIG. 1. Case I. (A) Well defined bone defect in lower left lambdoid suture. Also note elongated contour of dorsum sellae and posterior clinoid processes. (B) Half-axial view demonstrating the lambdoid bone defect with elevation and distortion of the left petrous bone. Note thinning and elongation of ipsilateral mandibular ramus and zygomatic arch.

shown) disclosed bilateral enlargement (L>R). A left cerebral arteriogram (Fig. 3B) after ventriculography showed right lateral ventricular hydrocephalus and a large mass filling the anterior third ventricle with downward displacement of the left ophthalmic artery. At surgery, a piloid astrocytoma was found involving both optic nerves, chiasm, the hypothalamus and region of the anterior third ventricle. A right Torkelson ventriculo-cisternostomy was performed followed by successful radiation



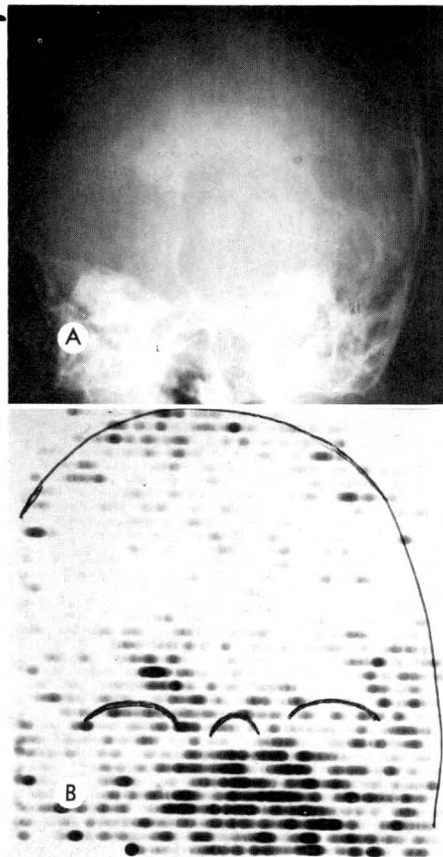
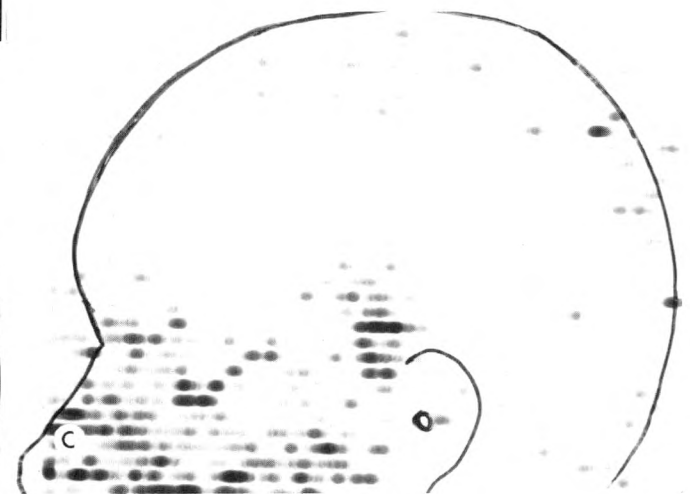
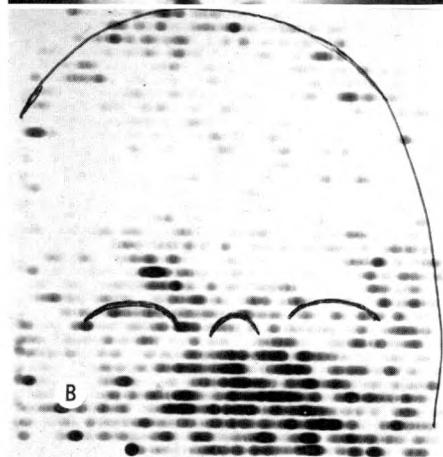


FIG. 2. Case II. (A) Roughly ovoid left lambdoid bone defect shown with post-mastoidectomy defect of left petrous bone. (B) Anterior projection of mercury 203 brain scan shows increased activity pattern just prior to death, over right brow ridge. (C) Right lateral projection of same scan locates tumor in region of right basal ganglia.



therapy. The patient was growing well and attending a school for the blind 2 years later.

CASE IV. N.W., Hosp. No. 58-16145. This was an 8 year old white male with recent decreased vision of his left eye, noted by his school nurse. The patient's mother had had a right forequarter amputation for neurosarcoma 10 years previously. A brother and 1 younger sister of a total of 5 siblings both bore marks of the condition. Following right tibial fracture at the age of 9 months and subsequent pseudarthrosis, the patient's right lower leg was amputated at the age of 5 years because of persistent osteomyelitis. Skull roentgenograms on admission showed a mildly "J" shaped sellar contour with anterior clinoid erosion. A pneumoencephalogram (Fig. 4) revealed the typical contour of chiasmatic cistern air beneath the tumor-involved optic chiasm.

Right frontal craniotomy disclosed that the optic nerves were enlarged to six times the nor-

mal size, with tumor extending into the chiasm and involving the hypothalamus. Biopsy diagnosis from the left optic nerve showed spongioblastoma polare. Radiation therapy followed. On follow-up, 5½ years later, the patient was doing well in school without recurrence of symptoms. Vision in right eye was 20/25 and in left eye 20/70.

CASE V. J.S., Hosp. No. 49-07657. This 32 year old white male with only a few café au lait spots of the trunk entered the hospital for physical rehabilitation following repeated surgical procedures. He was first noted to have left foot drop and pes cavus foot deformity at the age of 12 years. Progressive ataxia, hoarseness, hand incoordination, headache and bladder difficulties dated from the age of 21 years, at which time low thoracic and high cervical laminectomies were performed and three neurofibromata were removed. A spinal accessory neurofibroma was removed at this same time.

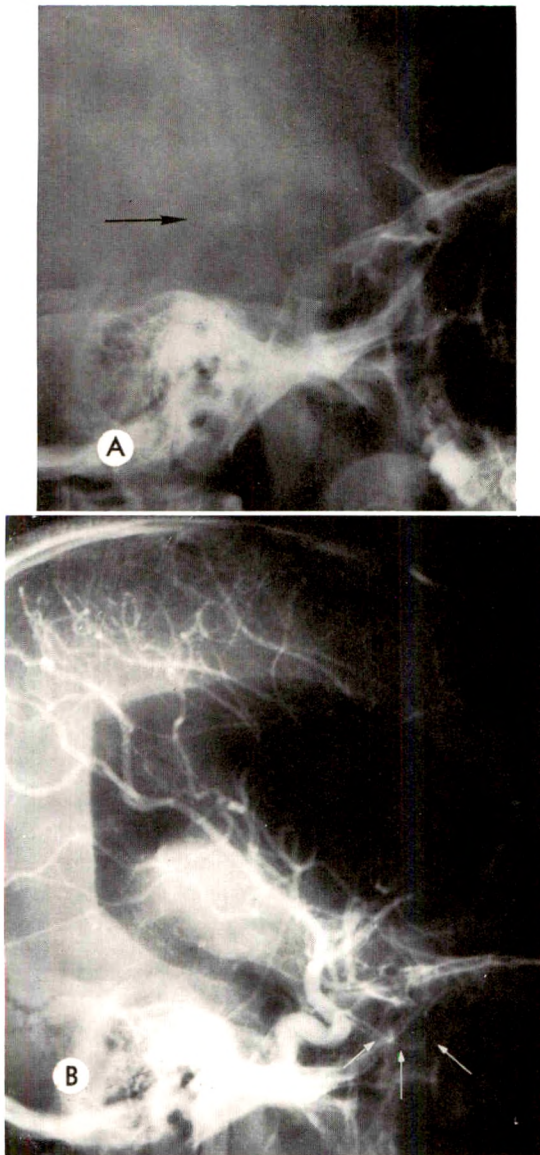


FIG. 3. Case III. (A) Lateral view of sella turcica showing "J" shape with concavity of chiasmatic groove, deepening of optic canal, erosion of dorsum sellae and posterior suprasellar tumor calcification (arrow). (B) Left lateral cerebral arteriogram immediately following ventriculography demonstrates the split coronal sutures and dilated right lateral ventricle due to a large tumor extending from the optic chiasm and obliterating the anterior third ventricle. The first portion of the left ophthalmic artery (arrows) is depressed by tumor extending down the expanded left optic canal before swinging upward to its more normal course under the orbital roof.

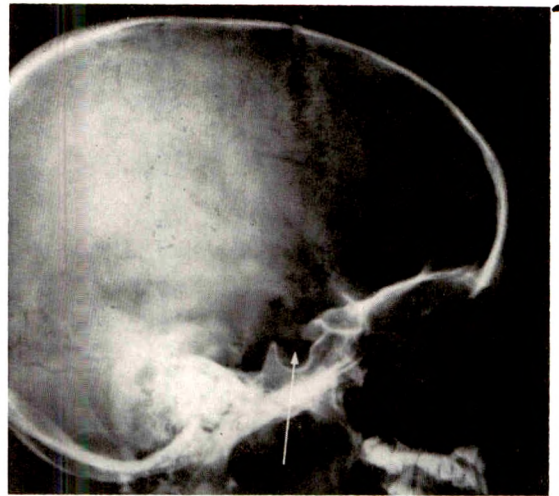


FIG. 4. Case IV. Mild "J" shaped contour of sella turcica is shown with erosion of the underside of the left anterior clinoid process. Crescent shaped air contour of the chiasmatic cistern underlies the optic chiasmal tumor which, according to Evans *et al.*,<sup>4</sup> is the most specific pneumoencephalographic sign of optic chiasmal glioma. Coronal suture diastasis gives evidence of increased intracranial pressure of some duration.

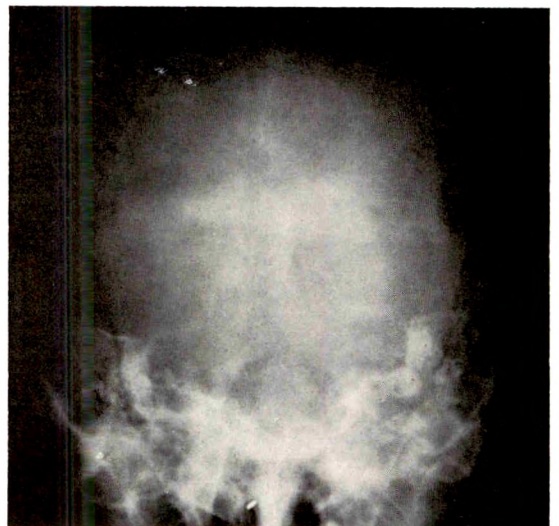


FIG. 5. Case V. Preoperative half-axial view shows extensive erosion of internal auditory canals and medial portions of both petrous bones due to bilateral acoustic tumors. Note silver clip on the right side near skull base where two high cervical neurofibromata and a spinal accessory neurofibroma were removed 8 years previously.



From the age of 27 to the age of 30 years, he experienced progressive hearing loss of the right ear, followed by hearing loss of the left ear. At surgery, a large right acoustic neurinoma was excised. Another tumor was seen attached to the meninges anterior to the medulla which could not be removed because of apnea which

occurred when traction was applied to the tumor. Postoperatively, following a 2 week period of coma, the patient recovered to the current alert status with persistent extremity paresis, marked ataxia, and deafness with glottic and vocal cord paralysis. A half-axial skull roentgenogram (Fig. 5) prior to removal of the

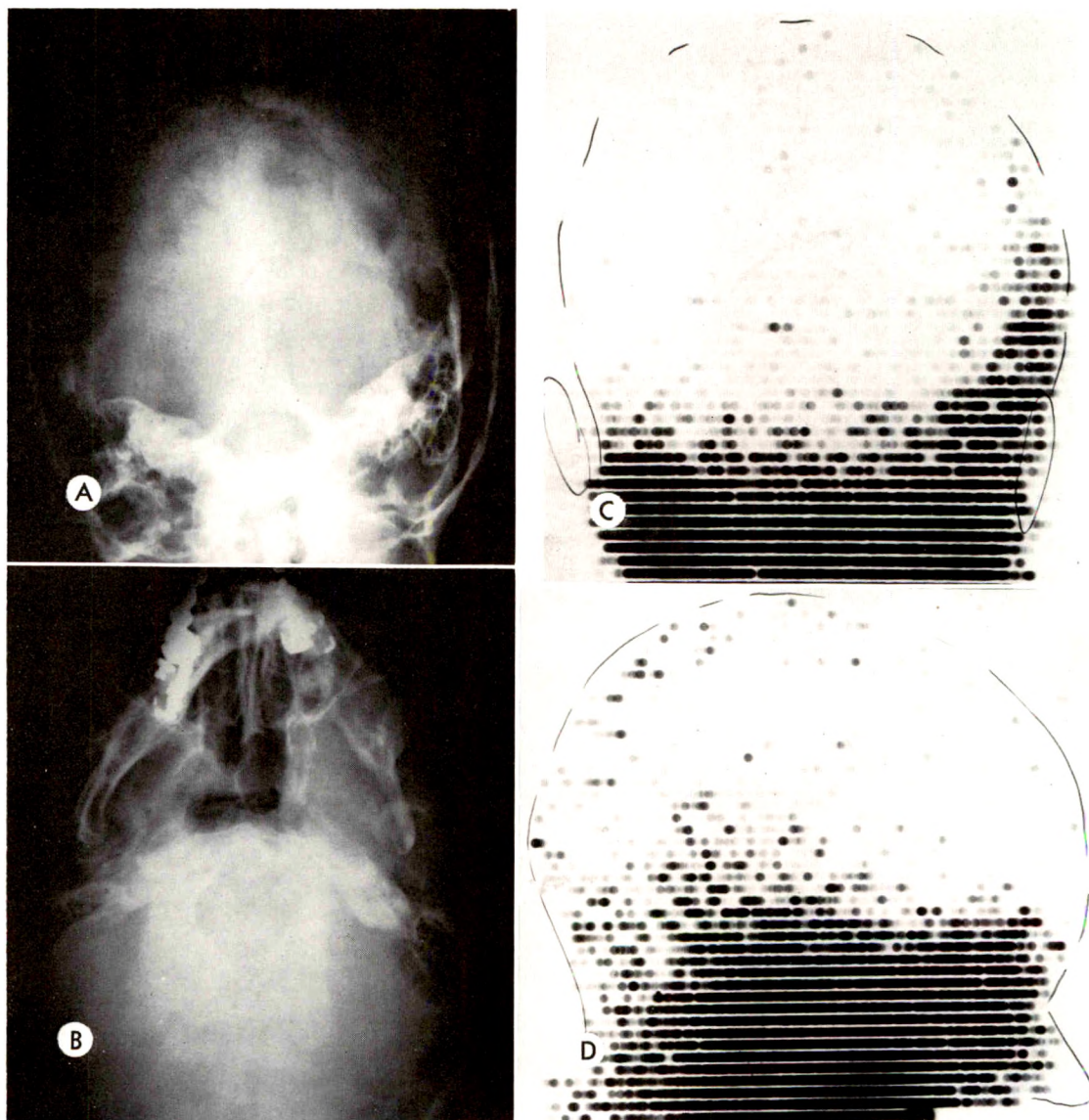


FIG. 6. Case VI. (A) Well defined left lambdoid bone defect with elevation and underdevelopment of ipsilateral petrous bone. (B) Submentovertical view showing mandibular deformity, left lambdoid bone defect and asymmetry of the middle fossae. The left greater sphenoid reflection (anterior boundary of middle fossa) is set further forward, but no orbital wall defect or exophthalmos was present. (C) Anterior projection of technetium 99m brain scan demonstrates the pattern of increased activity in the left anterior occipital region. (D) Left lateral scan shows that the activity area conforms to the lambdoid bone defect. No neurofibroma was present on examination in this area. Electroencephalogram and left cerebral arteriogram were interpreted as normal.

right acoustic neurinoma demonstrated petrous bone erosions due to bilateral acoustic neuromas. A mercury 203 brain scan and bilateral cerebral arteriograms showed no other evidence of tumor prior to the latest surgery at the age of 30 years.

CASE VI. S.L.K., Hosp. No. 65-23804. This 45 year old white married female was admitted for evaluation of a left post-auricular skull defect, first noticed by the patient on palpation of a tender area behind the left ear. The patient's father and brother showed marks of the disease. Observation of the patient showed numerous nodules and café au lait spots over her body. Cranial nerve examination was normal except for a right seventh nerve paresis. No central nervous system symptoms were elicited. A half-axial roentgenogram (Fig. 6A) showed a distinct left lambdoid suture defect with elevation and underdevelopment of the ipsilateral petrous bone. A submentovertical view (Fig. 6B) demonstrated mandibular deformity and deformity of the left greater sphenoid wing reflection (anterior wall of middle cranial fossa). Technetium 99m brain scans (Fig. 6, C and D) revealed an unexplained pattern of increased activity in the region of the left lambdoid bone defect. An electroencephalogram and left cerebral arteriogram were interpreted as normal.

#### DISCUSSION

The persons surveyed in this study represent approximately one-sixth of the 39 known patients with neurofibromatosis hospitalized between 1954 and 1966 at the Kansas University Medical Center. Efforts were made to locate other cases in the metropolitan Kansas City area for survey skull roentgenograms. Five more patients were found and skull roentgenograms were made. None of these showed skull changes. These additional cases had been seen previously for removal of plexiform neuromas and scoliosis problems.

Interestingly, 3 out of the 6 patients reported here had left lambdoid skull defects, a finding recently discussed by Joffe,<sup>6</sup> but apparently undocumented in the English literature before that time. Autopsy of 1 patient (Case II) revealed the lambdoid defect to be a local thinning of bone without

overlying or underlying tumor cyst, or other recognizable abnormality. This is essentially the same pattern seen with many other bony cystic and erosive appearing lesions in neurofibromatosis. One patient (Case VI) had an abnormal pattern of increased radioactivity in the area of the bone defect. This has not been previously recorded, to the author's knowledge. Again, other examinations failed to reveal an underlying tumor or explanation for the pattern of activity. Examples of right lambdoid suture defects could not be found in the material available.

Although they are more common, changes of the bony orbit were not included in this presentation. These changes are extensively discussed by Burrows.<sup>2</sup> However, some of the other deformities of the calvarium, facial bones, and mandible are illustrated (Cases I and VI). These distortions of bone width, length and contour can be diagnostic of neurofibromatosis, especially when more than one cranial bone is involved.

Enlargement or erosion of skull foramina due to expanding cranial nerve tumors is commonly found in neurofibromatosis (Cases III, IV and V). Especially is this true of the optic gliomas (the most common manifestation of the disease in children) and the acoustic neurinomas. The optic gliomas, as pointed out by Marshall,<sup>9</sup> may produce characteristic changes of the anterior sella turcica and optic canal due to regional pressure. This subject has been recently reviewed by Evans *et al.*<sup>4</sup> Contrary to earlier reports, suprasellar extensions of optic gliomas may occasionally show calcification. In younger patients, this tumor calcification may stimulate the more common craniopharyngioma. Lindgren and Di Chiro,<sup>8</sup> reporting on calcifying suprasellar tumors in children, found 3 of their series of 9 optic glioma cases with visible suprasellar calcification (note Case III).

Case II illustrates one of the less frequent intracranial tumors, the central gliomas. Other tumors, often slow growing, are found with this disease. Meningiomas (especially



of the posterior fossa and often multiple) and other rarer tumors have been reported.<sup>10</sup> Arteriography, gas encephalography, and isotopic scanning procedures may be especially helpful in localizing these various tumors, found particularly in the middle years of life. As noted by others,<sup>1,3</sup> neurofibromatosis patients with central nervous system disease usually show few or none of the classic cutaneous signs of the disease. Also, it is unlikely that the well marked neurofibromatosis patient would have any of the central nervous system tumors.

Tumors within the spinal cord are less common than tumors of the nerve roots of the cord in this condition. However gliomas, especially astrocytomas and ependymomas of the cord are reported. Meningoceles, especially of the dorsal and lumbar regions, are fairly common. Case v demonstrated the frequent association of neurofibromas of the cord nerve roots with bilateral acoustic nerve tumors. These same people not infrequently have multiple meningiomas of the posterior fossa and of the spinal cord sheath.

#### SUMMARY

Six patients are reported illustrating some of the skull and intracranial manifestations of neurofibromatosis. These lesions, consisting of skull contour changes and various skull bone defects, are sometimes associated with a variety of intracranial tumors.

Neurofibromatosis patients with central nervous system tumors usually show few or none of the classic cutaneous marks of the disease.

The cases reported here further support

the interesting observation of a distinct roentgenologic sign in neurofibromatosis: the local calvarial defect of the left lambdoid suture.

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## NEUROFIBROMATOSIS\*

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NEUROFIBROMATOSIS is a congenital and familial<sup>37</sup> disease with widespread manifestations, such as lesions of the skin, tumors of the central and peripheral nervous system, and abnormalities of bone. The diagnosis is usually obvious when there are multiple cutaneous neurofibromas associated with pigmented lesions (café au lait spots). These characteristic lesions, however, are not always present, and may be so overshadowed by bizarre bone and soft tissue manifestations that the clinical diagnosis may be difficult.

### PATHOGENESIS

Neurofibromas develop from nerve sheaths, but there is disagreement as to the cellular origin. The problem is whether neurofibromas develop from the neuroectodermal sheath cells of Schwann, or from the mesodermal connective tissue elements of the nerve sheath, or both. Saphir<sup>42</sup> feels that both origins are likely because of the marked variation of the histologic pattern in neurofibromatosis.

Since it is difficult to explain the numerous bone abnormalities on the basis of involvement of the nervous system, it is believed that there is an associated mesodermal dysplasia, as noted by Holt and Wright<sup>21</sup> and emphasized by Hunt and Pugh.<sup>22</sup>

Other abnormalities described in association with neurofibromatosis and which are difficult to explain include osteomalacia,<sup>13</sup> endocrine abnormalities,<sup>25</sup> and vascular involvement.<sup>16</sup>

### SKIN

The typical skin lesions of neurofibromatosis are soft skin nodules (fibroma moluscum). These vary considerably in size, and may be sessile or pedunculated. At times, growth may be rapid. Remission also

occurs. Sarcomatous change develops in 5 per cent<sup>22</sup> to 10 per cent.<sup>38</sup>

The skin nodules are frequently associated with areas of pigmentation. These pigmented areas (café au lait spots) are brownish macules which vary considerably in size. They usually have smooth borders, which distinguishes them from the café au lait spots of fibrous dysplasia, which tend to have irregular ragged margins. When the classic skin lesions are present, the clinical diagnosis is obvious, but skin lesions are not always present.

The skin nodules projecting over the chest may cast soft tissue shadows simulating pulmonary metastases. Their soft tissue location can be established by roentgenologic examination in multiple projections, or by painting them with barium paste.<sup>25</sup> The roentgenologic appearance of the soft tissue nodules has been referred to as the "button sign" by Miller *et al.*<sup>33</sup>

### GASTROINTESTINAL TRACT

Neurogenic tumors of the gastrointestinal tract are rare. We have observed 1 case of gastric neurofibrosarcoma and 2 cases of neurofibroma of the small bowel. None of these cases had evidence of systemic neurofibromatosis.

In a study of 1,399 cases of benign tumors of the small bowel, River *et al.*<sup>39</sup> found neurogenic tumors in 6.5 per cent. Of these, only 17 per cent had cutaneous manifestations of neurofibromatosis. Of 57 cases in which the location was specified, the tumor was extraluminal in 25, intraluminal in 18, intramural in 6, and combined in 7.

The symptoms and findings listed by River *et al.*, in order of decreasing frequency, included pain, hemorrhage, nausea, vomiting, mass, distention, constipation, diarrhea, weight loss and cachexia.

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FIG. 1. Gastric neurofibrosarcoma. Large submucosal mass, with sharp margins. Mucosa intact.

nant. They are indistinguishable from other extramucosal tumors. They present as sharply outlined submucosal or intraluminal filling defects. This is well demonstrated by our case of neurofibrosarcoma of the stomach (Fig. 1).

If the tumor grows outward (subserosal), it may be more difficult to visualize. Its presence may be detected only by extrinsic pressure on adjacent loops of small bowel, as demonstrated by one of our cases of neurofibroma of the small bowel (Fig. 2). This separation of loops of small bowel has been referred to as the "blank space" effect by Good.<sup>12</sup>

Manifestations of obstruction, volvulus or intussusception may obscure the primary lesion. Intussusception was present in 1 of our cases of neurofibroma of the small bowel (Fig. 3).

Ulceration is not unusual. If the ulcer is



FIG. 2. Jejunal neurofibroma in a 48 year old woman. Extraluminal pedunculated tumor,  $3 \times 1.5$  cm. Pressure on adjacent loops of small bowel resulting in "blank space" effect. (Reproduced with permission from *Postgraduate Medicine*.<sup>40</sup>)

Complications were intestinal obstruction, intussusception and volvulus.

The roentgenologic findings have been summarized by River and Schorsch<sup>40</sup> and by Marshak *et al.*<sup>29</sup> Neurogenic tumors may be single or multiple, benign or malig-

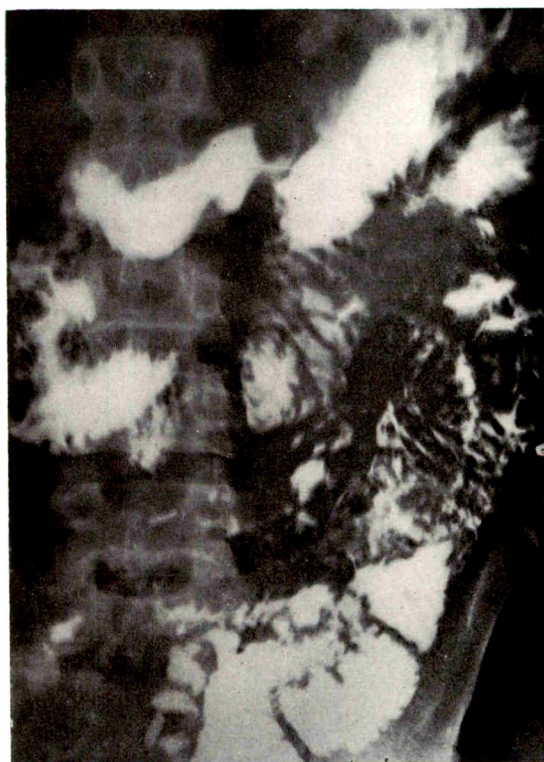


FIG. 3. Jejunal neurofibroma in a 45 year old man. "Coil-spring" effect near ligament of Treitz due to intussusception. (Reproduced with permission from *Postgraduate Medicine*.<sup>40</sup>)



irregular and unusually large, sarcomatous change should be suspected.<sup>29,31</sup>

An unusual manifestation of neurofibromatosis of the colon was recently described by Staple *et al.*<sup>44</sup> There was plexiform neurofibromatosis of the colon which simulated Hirschsprung's disease. Roentgenologically, the rectum and distal sigmoid were somewhat narrowed, whereas the remainder of the colon was dilated and atonic. Histologically, there was an increase in size and number of the nerves of the myenteric and submucosal plexuses. Generalized neurofibromatosis was present, with multiple subcutaneous masses and gigantism of the right lower extremity.

#### CHEST

In neurofibromatosis there are 3 causes of a thoracic mass: (1) "dumbbell" neurofibroma, (2) intercostal neurofibroma or (3) intrathoracic meningocele.

*"Dumbbell" Neurofibroma.* The mass is

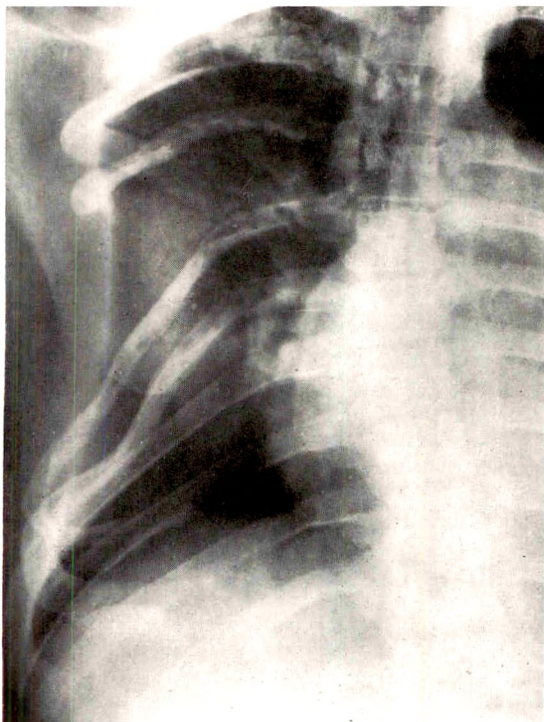


FIG. 4. Intercostal neurofibroma in a 32 year old man. Lateral intrathoracic mass with extrinsic pressure changes on ribs.

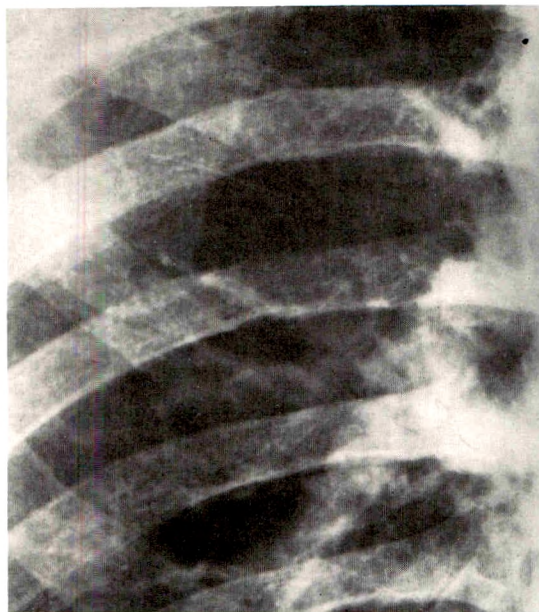


FIG. 5. Rib notching due to intercostal neurofibromatosis.

located in the posterior mediastinum. It arises in the spinal canal and extends into the chest by way of the intervertebral foramen. Its intraspinal origin may be demonstrated by roentgenograms of the thoracic spine. There is widening of the intervertebral foramen. Pressure defects may be noted on the pedicles, vertebral body, or posterior ribs.

*Intercostal Neurofibroma.* Occasionally, a neurofibroma arises from the peripheral portion of an intercostal nerve. In such cases, the tumor is not located in the posterior mediastinum. The lesion may extend into the chest, simulating a lung tumor. Extrinsic pressure defects on the adjacent ribs testify as to the long duration of the intercostal neurofibroma (Fig. 4 and 5).

*Intrathoracic Meningocele.* Another cause of a thoracic mass in the presence of neurofibromatosis is an intrathoracic meningocele<sup>30</sup> (Fig. 6, A and B). Three-fourths of reported cases of intrathoracic meningocele have been associated with neurofibromatosis.<sup>8</sup> If a patient with neurofibromatosis has a thoracic mass, a meningocele is more likely than a neurogenic tumor.<sup>6</sup>



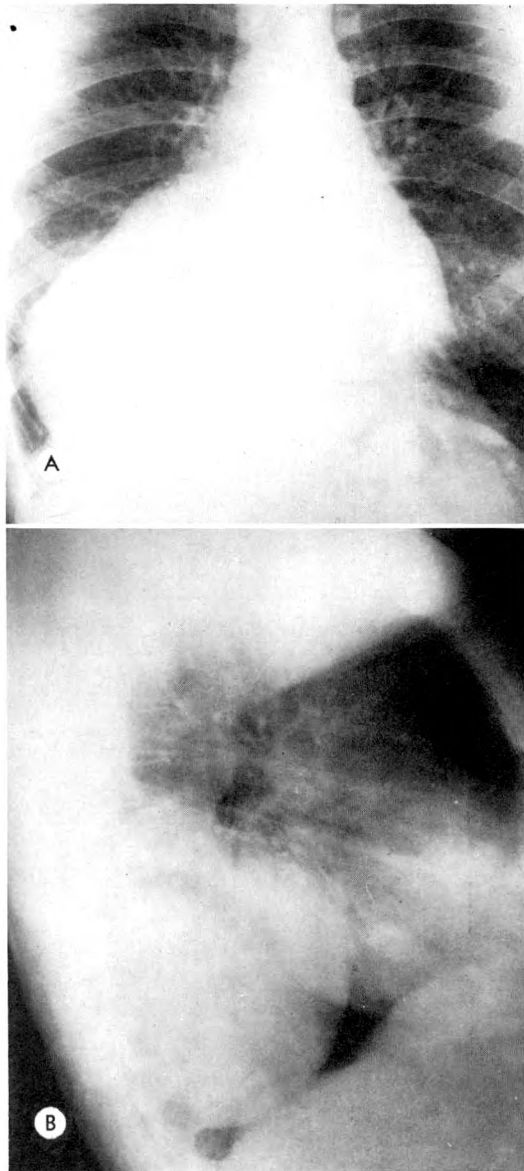


FIG. 6. Intrathoracic meningocele. (A) Large mass in the lower right chest. (B) Posterior location. Intervertebral foramina are widened.

The lateral thoracic meningocele varies considerably in size. It may be massive, filling most of the hemithorax. In other cases, meningoceles are so small that their outline is not visualized on the roentgenogram. Their presence may be suspected, however, if several intervertebral foramina are enlarged, since multiple foraminal enlargement is more often due to menin-

gocles than neurofibromatous tumors.<sup>28</sup>

The intrathoracic meningocele extends into the chest through an intervertebral foramen in the same manner as a "dumb-bell" neurofibroma. On plain roentgenograms, therefore, an intrathoracic meningocele is indistinguishable from an intrathoracic neurofibroma. There is a posterior mediastinal mass, with associated intervertebral foraminal widening, and pressure defects on the pedicles, vertebral bodies, and posterior ribs.

The presence of a meningocele is confirmed by myelography, which demonstrates communication between the subarachnoid space and the intrathoracic mass. Because of the large volume of contrast material necessary to opacify the mass, gas myelography is preferable to opaque contrast material.<sup>3,20,28</sup>

#### BONE CHANGES

The bone changes in neurofibromatosis have been documented in the classic de-



FIG. 7. Twenty-three year old man with erosion and invasion of the medial aspect of the left pelvis due to neurofibrosarcoma of adjacent soft tissues.

scription of Holt and Wright.<sup>21</sup> The incidence of bone involvement has been reported as high as 29 per cent<sup>21</sup> and 51 per cent.<sup>22</sup> The true incidence of bone abnormality is difficult to state, since it varies with the extent of the roentgenologic examination.

Roentgenologic survey of all bones is advised in patients with neurofibromatosis. This will result in a better appreciation of the incidence of bone changes. It is also believed that more bone abnormalities will be detected in asymptomatic locations. Furthermore, there will be a better understanding of the natural history of the lesions.

#### KYPHOSCOLIOSIS

Kyphoscoliosis is one of the most characteristic osseous abnormalities of neurofibromatosis (Fig. 8; and 9, *A* and *B*). Scoliosis is present in 10 per cent of patients with neurofibromatosis.<sup>23</sup> Scoliosis has been found in up to 43 per cent of patients with neurofibromatosis who have bone abnormalities.<sup>32</sup>

A mild and non-characteristic scoliosis may occur if there is difference in the length of the lower extremities due to growth dis-

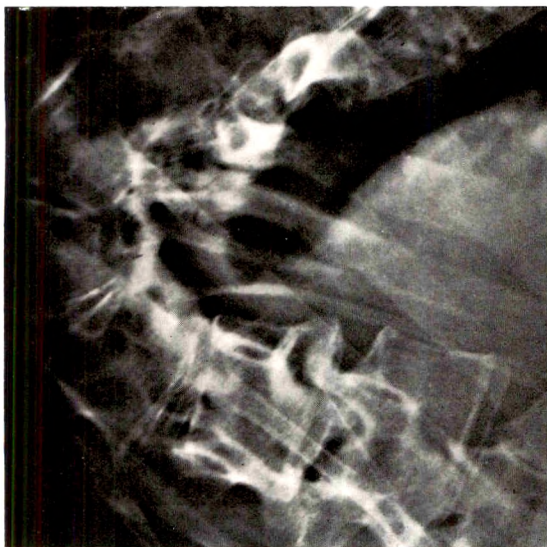


FIG. 8. Angular scoliosis which caused paraplegia in a 27 year old woman. Some ribs show an attenuated "twisted-ribbon" appearance.

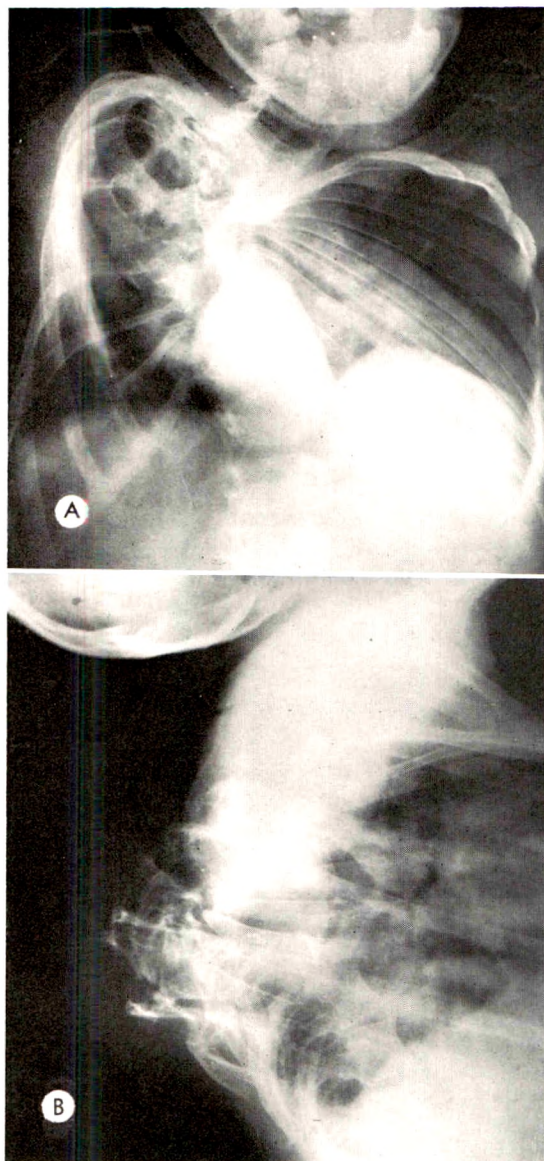


FIG. 9. Scoliosis in an 8 year old girl. (*A*) Sharp angular thoracic scoliosis with dysplastic rib deformities. (*B*) Extreme kyphosis. Overlying soft tissue mass is a neurofibroma.

turbance. This should not be confused with the characteristic type of scoliosis which occurs in neurofibromatosis.

The typical scoliosis of neurofibromatosis is very acute and angular, extending over a relatively short distance, as contrasted to the long smooth curve of idiopathic scoliosis. The lower thoracic region is most commonly involved, but in some cases the



deformity occurs in the cervical or lumbar region. Kyphosis often predominates over scoliosis. The deformity may be so severe that paraplegia develops.<sup>32</sup>

The pathogenesis of the scoliosis is obscure. Heard *et al.*<sup>17</sup> listed 5 possible etiologic factors: (1) neurofibromatous infiltration, (2) extrinsic pressure erosion from local tumor, (3) osteomalacia, (4) osteoporosis due to muscular weakness, and (5) mesodermal dysplasia.

Although neurogenic tumors have been found at the site of scoliosis,<sup>7,11</sup> in many cases neurofibromatous tissue is absent at surgery.<sup>22</sup> For this reason the mesodermal dysplasia theory is attractive. This etiology is also suggested by the bizarre appearance of the vertebrae, which may be so deformed that their individual contours are scarcely recognizable. The mesodermal dysplasia theory, however, fails to explain the absence of similar deformities of the long bones (unless congenital bowing is considered analogous).

The presence of a meningocele has been noted in the region of vertebral scoliosis.<sup>6,20</sup> This cannot be a constant causative factor, since a meningocele is not always present.

As a significant factor producing scoliosis, we believe that there is vertebral weakening due to pressure erosion from a dilated dural sac. In 1 case of scoliosis, we observed diffuse dilatation of the dural space, plus multiple small lateral meningoceles. Loop *et al.*<sup>28</sup> have demonstrated meningeal dilatation at the site of scoliosis, without associated meningocele formation. Additional myelographic studies are needed to confirm the constancy of these findings.

#### PRESSURE EROSION OF BONE

Neurofibromas erode adjacent osseous structures by pressure. This has been referred to as the "pit" or "cave" appearance.<sup>34</sup> These lesions are typically noted in long bones. In advanced cases, neurofibromas extend into and replace bone (Fig. 7), and may produce osseous deformity.<sup>15</sup>

In the skull, pressure erosion may cause widening of the foramina. In the spine,

widening of the intervertebral foramina is a familiar manifestation. Associated pressure defects are commonly noted on the pedicles and vertebral bodies.

There are several types of rib involvement. Erosion of a rib near its vertebral margin occurs with a "dumbbell" neurofibroma or lateral meningocele. Rib erosion in other locations occurs due to neurofibroma of a distal portion of an intercostal nerve (Fig. 4). Occasionally, multiple intercostal neurofibromas produce "notching" of the inferior rib margins similar to coarctation of the aorta (Fig. 5). Another deformity of the ribs has been described as the "twisted ribbon" appearance by Holt and Wright (Fig. 8). The ribs are thin and elongated, and scoliosis is frequently but not invariably associated. The "twisted ribbon" appearance may be explained by local pressure erosion,<sup>21</sup> but since some of these patients do not have intercostal neurofibromas,<sup>22</sup> a dysplastic etiology is most likely.

#### OVERGROWTH OF BONE

Overgrowth may occur in the skull, mandible or pelvis, but the extremities are most frequently involved (Fig. 10). Any or all of the extremities may be affected, often resulting in gigantism.

Overgrowth of bone is usually associated with elephantiasis-like enlargement of the



FIG. 10. Bone overgrowth in a 14 year old boy. Bones are elongated and slender. Elephantoid hypertrophy of the soft tissues.

overlying soft tissues (Fig. 10). The soft tissue mass contains a diffuse (plexiform) neuroma, as well as hemangiomatous and lymphangiomatous elements.

On roentgenologic examination, the affected extremity is increased in length. The bones may show a sinuous "S"-shaped tortuosity. Irregular periosteal thickening occasionally occurs.

The bones may be either increased or decreased in width. Combinations of hyperplasia and hypoplasia occur, resulting in distorted growth.

Overgrowth of bone has been explained as being due to an increased blood supply,<sup>21,22</sup> but mesodermal dysplasia may also be a factor.

#### HYPOPLASIA OF BONE

Hypoplasia of bone may be observed as a decrease in width. This may be present even though the bone is increased in length.

The hypoplastic "twisted ribbon" appearance of the ribs has been mentioned above.

An interesting type of hypoplasia occurs in the skull of patients with neurofibromatosis. This is manifested as an absence of the posterosuperior wall of the orbit, and is associated with exophthalmos. Generalized neurofibromatosis may or may not be present. The defect was first described in 1933 by LeWald<sup>26</sup> in 4 patients, 2 of whom had neurofibromatosis. Hunt and Pugh<sup>22</sup> found orbital defects in 7 per cent of 192 patients with neurofibromatosis. In 6 cases of congenital unilateral exophthalmos, Bruwer and Kierland<sup>4</sup> found evidence of neurofibromatosis in 5. Although we have seen only 1 patient with this abnormality, it has been observed with sufficient frequency to be considered a characteristic osseous manifestation of neurofibromatosis.<sup>22</sup> Since neurofibromatous tissue may not be present at surgery, Hunt and Pugh feel that this defect is due to mesodermal dysplasia, rather than to localized pressure erosion.

Roentgenologically, there is an absence of bone in the posterosuperior wall of the orbit. The orbital defect also involves both wings of the sphenoid and a portion of the

frontal bone. Other findings include enlargement of the orbit, elevation of the sphenoid ridge, deformed or absent clinoid processes and enlargement of the middle cranial fossa.<sup>4,43</sup>

An unusual type of hypoplasia is "cranium bifidum,"<sup>22</sup> a large defect in the posterior aspect of the skull adjacent to the midline.

A special type of osseous hypoplasia is manifested by congenital bowing and pseudarthrosis.

#### CONGENITAL BOWING AND PSEUDARTHROSIS

Bowing and pseudarthrosis usually occur together. Generalized neurofibromatosis may or may not be present. The association of pseudarthrosis with neurofibromatosis was first observed by Ducroquet<sup>10</sup> who reported 11 patients, 9 of whom had neurofibromatosis.

Bowing of the tibia and fibula is usually anterior and may be present at birth. Pseudarthrosis usually develops at the middle or lower third of the tibia and/or fibula, and may occur after fracture, osteotomy or spontaneously.

Roentgenographically, the ends of the bone are rounded and smooth. There is a "pencil-point" appearance of the tibia or fibula due to tapering and pointing of the bone ends. In some cases, the bone ends are considerably separated, due to hypoplasia of bony tissue (Fig. 11A).

Green and Rudo<sup>14</sup> described the presence of intra-osseous neurofibromatous tissue at the site of the pseudarthrosis. Usually, however, these histologic findings are not demonstrated. Aegerter,<sup>1</sup> for instance, examined 15 cases and found only masses of fibrous tissue. More recently, Aegerter and Kirkpatrick<sup>2</sup> stated that the fibro-osseous tissue interposed between the bone ends is the result and not the cause of the abnormality. They concluded that defective bone formation was the basic cause of the pseudarthrosis. This correlates well with the frequent roentgenologic evidence of localized absence of bone in the fibula in association with tibial pseudarthrosis.



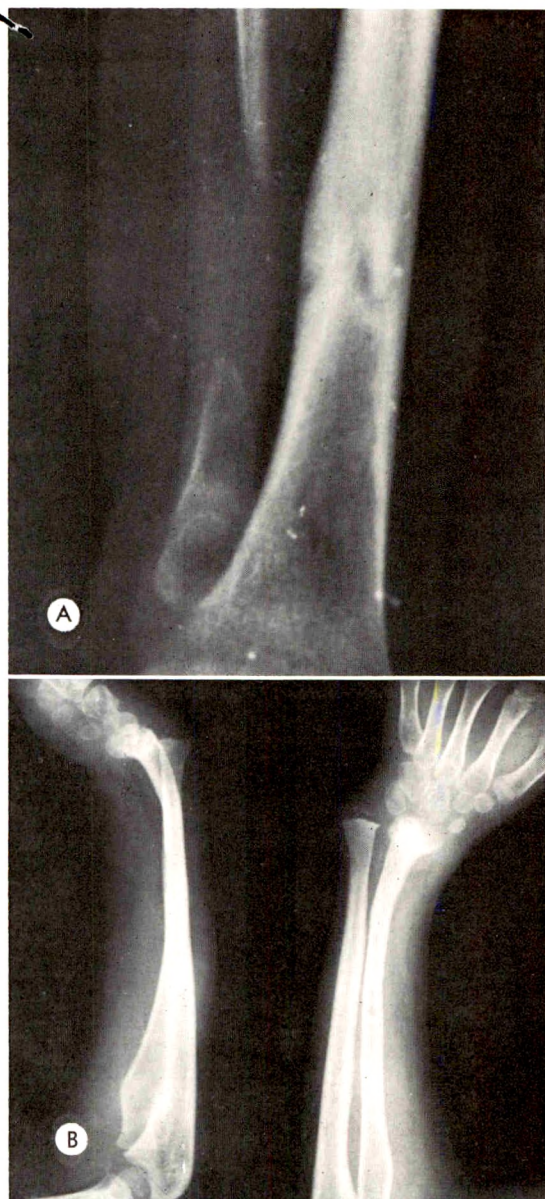


FIG. 11. (A) Pseudarthrosis involving the tibia. Marked hypoplasia of fibula, with "pencil-point" tapering. (B) Madelung's deformity of the forearm in the same patient, an 11 year old girl.

#### INTRA-OSSEOUS RADIO- LUCENT LESIONS

Benign intra-osseous "cystic-like" radiolucent lesions due to neurofibromatous tissue in bone have been described by several authors.<sup>3, 9, 11, 14, 15, 21</sup> Documentation has varied from good<sup>9</sup> to incomplete.<sup>3</sup> The existence of this type of lesion must be ac-

cepted in spite of its infrequency. A malignant intra-osseous neurogenic tumor also occurs,<sup>35</sup> but is even more uncommon.

In addition to neurofibromatous tissue within bone, there are several other circumstances in which there appears to be an intra-osseous radiolucency.

An intra-osseous lesion may result from the deposition of a periosteal shell of bone around a lesion originally external to the bone.<sup>3, 19, 21, 30</sup> The result is a periosteal neurofibroma ("subperiosteal bone cyst") which protrudes outward and is not mainly intra-osseous.

In some circumstances, an intra-osseous defect may be more apparent than real. For instance, an extrinsic pressure defect on the margin of a bone may simulate an intra-osseous radiolucency, unless the lesion is studied in 2 projections (Fig. 13B). Alteration of the trabecular pattern in association with bowing may simulate the margins of an intra-osseous defect.

A non-osteogenic fibroma may be present as an incidental finding, and should not be mistaken for a neurofibromatous lesion.

Hunt and Pugh<sup>22</sup> suggested osseous dysplasia as a cause of intra-osseous radiolucent lesions.

Osseous radiolucencies could be explained by considering fibrous dysplasia to be in some way related to neurofibromatosis,<sup>1, 45</sup> but it is generally felt that these conditions are separate entities.<sup>27</sup>

In spite of the several possible causes, intra-osseous radiolucent lesions are quite rare.

#### BONE SCLEROSIS

Sclerosis of the bone is rare, but does occur in neurofibromatosis. McCarroll<sup>30</sup> found sclerosis in 2 cases, which he interpreted as "melorheostosis." In both cases there was overlying soft tissue hypertrophy. Friedman<sup>11</sup> also reported a case with sclerosis. Disordered growth was present, with hypoplastic features predominating. There was cortical thickening, coarse linear medullary sclerosis, and cyst-like areas.

We have observed a case of sclerosis,



associated with hypertrophy of the overlying soft tissues. The sclerosis affected the clavicle, scapula, humerus, radius and ulna. Cortical thickening and sclerosis were the most prominent findings, but medullary sclerosis was also present. In some of the sclerotic areas there was a definite "smudged" appearance similar to that observed in fibrous dysplasia (Fig. 13, *A* and *B*).

We believe that osseous sclerosis is a manifestation of osseous dysplasia occurring in neurofibromatosis.

#### CONGENITAL BONE ABNORMALITIES

There is an increase in the frequency of congenital osseous abnormalities in patients with neurofibromatosis. Hunt and Pugh<sup>22</sup> found 33 abnormalities in a series of 192 patients. Most of these were unimportant, such as spina bifida, sacralization and intertrib fusions.

Spondylolisthesis was found in 4 cases by McCarroll and in 2 cases by Hunt and Pugh, but there were no illustrations. We have observed 1 case of spondylolisthesis, consisting of anterior and lateral displacement of the 4th lumbar vertebra (Fig. 12, *A* and *B*). There was a large neurofibroma

in this region, which is believed to be responsible for the spondylolisthesis. There was no evidence of a congenital defect in the pars interarticularis.

In 1 of our cases there was a typical "Madelung's" deformity of the distal radius and ulna (Fig. 11*B*). (Miller *et al.*<sup>33</sup> previously reported the presence of Madelung's deformity in this patient, but did not include a roentgenologic illustration.)

#### CENTRAL NERVOUS SYSTEM

Neurofibromas of the central nervous system are often multiple.

Neurofibromas of the cranial nerves are not uncommon. Acoustic neuromas may produce enlargement of the internal acoustic meatus. Optic nerve tumors may result in widening of an optic foramen.

Neurofibromas arising from the spinal canal usually arise as extradural tumors from the root sheaths of the spinal nerves. They often extend through the intervertebral foramen, with pressure defects on the intervertebral foramen, vertebrae, and adjacent ribs. Neurofibromas may also arise intradurally. These produce early symptoms of cord compression, and seldom produce changes in ordinary roentgenograms.

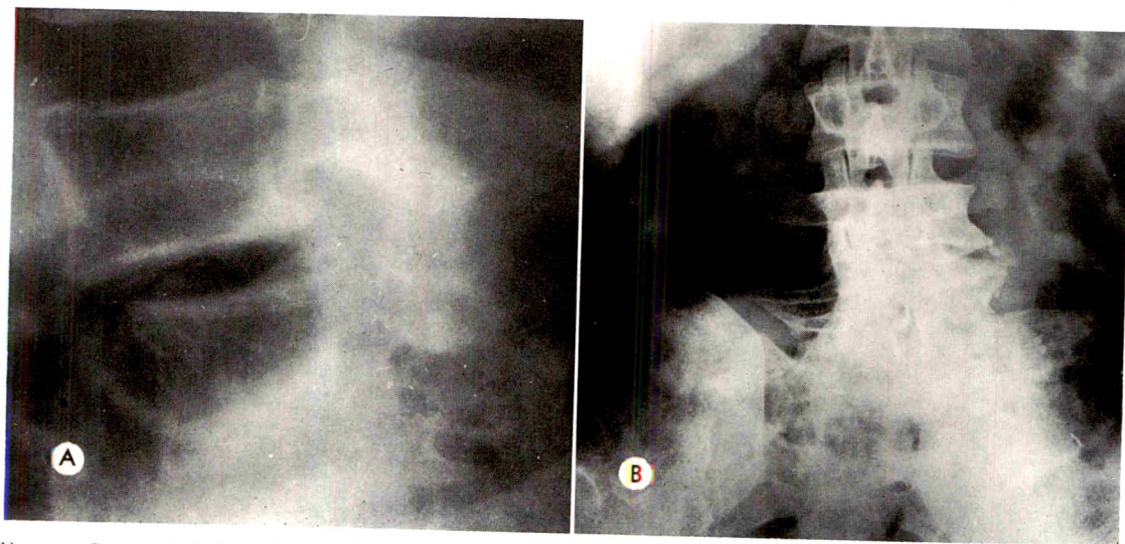


Fig. 12. Spondylolisthesis in a 59 year old man. (*A*) Anterior displacement of the fourth lumbar vertebra. No defect in the pars interarticularis. (*B*) Frontal view demonstrating displacement of the fourth lumbar vertebra to the left due to a large neurofibromatous soft tissue mass in the lower right lumbar region. Extrinsic pressure defects on the lower lumbar vertebrae and right transverse processes.



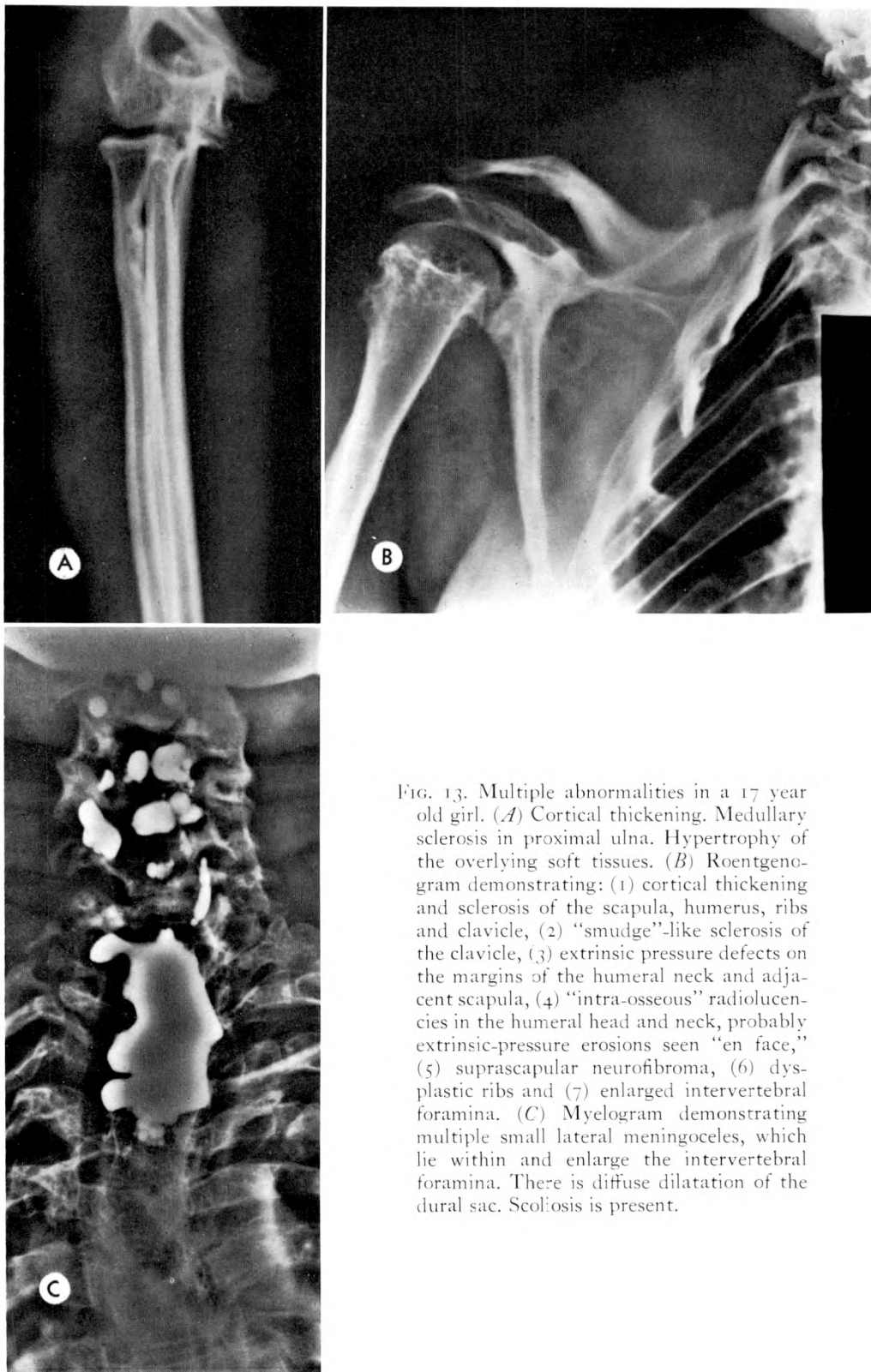


FIG. 13. Multiple abnormalities in a 17 year old girl. (A) Cortical thickening. Medullary sclerosis in proximal ulna. Hypertrophy of the overlying soft tissues. (B) Roentgenogram demonstrating: (1) cortical thickening and sclerosis of the scapula, humerus, ribs and clavicle, (2) "smudge"-like sclerosis of the clavicle, (3) extrinsic pressure defects on the margins of the humeral neck and adjacent scapula, (4) "intra-osseous" radiolucencies in the humeral head and neck, probably extrinsic-pressure erosions seen "en face," (5) suprascapular neurofibroma, (6) dysplastic ribs and (7) enlarged intervertebral foramina. (C) Myelogram demonstrating multiple small lateral meningoceles, which lie within and enlarge the intervertebral foramina. There is diffuse dilatation of the dural sac. Scoliosis is present.

Patients with neurofibromatosis show an increased incidence of other types of tumors, such as gliomas and meningiomas.

**Myelography.** Neurofibromatous tumors may produce one or more filling defects in the myelogram. Another finding, which is less well known, is generalized dilatation of the subarachnoid space. This dilatation of the dural sac may occur without the presence of neurofibromatous tumors, and is thought to be due to mesodermal dysplasia involving the meninges.<sup>24,28,41</sup> This correlates with the opinion of Turner and Gardner,<sup>46</sup> who felt that neurofibromatosis might represent a defect in the supporting or binding tissues of the nervous system.

We have observed concave defects on the posterior vertebral margins in the absence of tumors, as have others.<sup>6,18,20,28,41</sup> It should be emphasized that these posterior vertebral defects are produced by anterior sac-like protrusions of the dilated dural sac (Fig. 14), and may occur *without* the presence of meningoceles or neurofibromas.

Meningoceles are produced by lateral sac-like protrusions of the dilated dural sac (Fig. 13 C). These meningoceles protrude through the intervertebral foramina. A meningocele may produce enlargement of an intervertebral foramen *without* the presence of a localized neurofibroma, as we have observed roentgenographically. This has been demonstrated surgically by Loop *et al.*<sup>28</sup> If multiple intervertebral foramina are enlarged, meningoceles are more likely to be present than neurofibromatous tumors.<sup>28</sup>

In neurofibromatosis, meningoceles are most common in the thoracic area, but may also occur in the cervical,<sup>17</sup> lumbar<sup>41</sup> or sacral<sup>22</sup> regions.

#### SUMMARY AND CONCLUSIONS

Neurofibromatosis is a dysplasia which involves mesodermal as well as ectodermal elements. This results in a broad spectrum of roentgenologic findings.

Three cases of neurofibromatous tumors of the gastrointestinal tract are illustrated.

An example of bone sclerosis is documented.

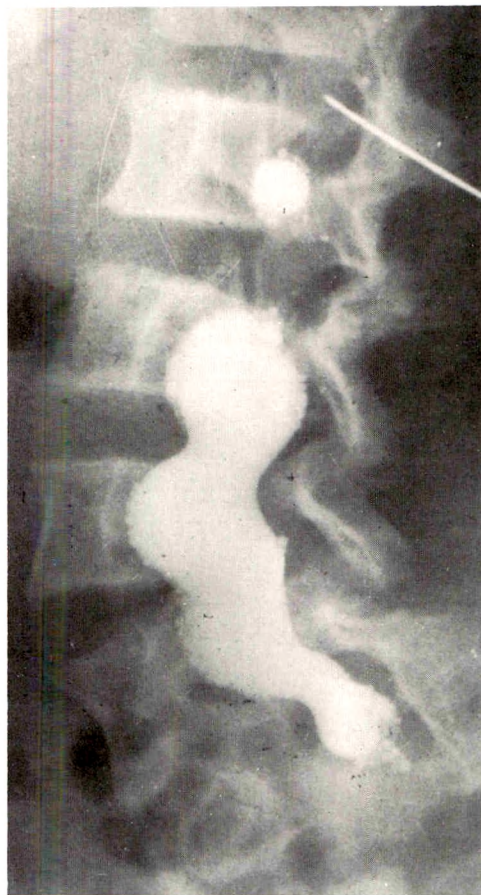


FIG. 14. Myelogram of a boy with dorsal vertebral excavations. These excavations are produced by anterior sacculations of the dilated dura. Note absence of neurofibromatous tumor.

Dilatation of the dural sac is illustrated and emphasized. Meningoceles, widening of intervertebral foramina, and erosion of posterior vertebral margins may be produced by the dilated dural sac, without the presence of neurofibromatous tumors.

We believe that vertebral weakening due to pressure from the dilated dural sac is a significant factor in the production of scoliosis.

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## CEREBRAL LEPTOMENINGEAL CYSTS OF DEVELOPMENTAL ORIGIN\*

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**I**NTRACRANIAL leptomeningeal (arachnoidal) cysts are collections of usually clear fluid enclosed by thin membranes. The term leptomeningeal indicates the resemblance of the membranous covering to normal arachnoid and also describes the location of the cyst in relation to the meninges. Leptomeningeal cysts occur below<sup>1, 8, 12, 15, 26</sup> and above<sup>2, 4, 5, 6, 7, 16, 19, 21, 22, 25, 27</sup> the tentorium, within the subarachnoid cisterns,<sup>10, 14</sup> and between the cerebral hemispheres.<sup>13</sup> Omitting those cases clearly associated with fractures of the calvarium, the relationship of leptomeningeal cysts to trauma, infection, and developmental defects has been documented in few instances. A unitary theory of origin is presently unacceptable, and an etiologic classification of leptomeningeal cysts based upon a review of the literature would be more confusing than useful.

The purpose of this report is to present the roentgenologic features of a cerebral leptomeningeal cyst, the probable developmental origin of which was established by operation. The uncertain origin of most nontraumatic leptomeningeal cysts justifies the reporting of individual cases having clinical, roentgenologic and anatomic documentation.

### REPORT OF A CASE

U.H. 04-59-25-5. A 26 year old female entered the University of Kentucky Hospital on September 17, 1965 with a chief complaint of episodic left temporal headaches accompanied by diplopia, right hemiparesis, and numbness of the right side. Six episodes had occurred during the previous 2 years, the last 3 weeks before admission. For at least 1 year, she had dropped objects from her right hand and had become aware of slight weakness in the right arm and



FIG. 1. Frontal roentgenogram of skull showing thinning in the fronto-parietal area.

hand. She denied head trauma and any illness involving the nervous system in the past.

Examination of the patient disclosed mild weakness of the right upper extremity and slight facial asymmetry. Reflexes in the right arm were questionably hyperactive. Appreciation of all sensory modalities was reduced over the entire right side of her body. The remainder of the physical examination was normal.

Basic laboratory studies were normal. Plain roentgenograms of the skull (Fig. 1) revealed a large area of thinning involving the inner table and diploe in the left fronto-parietal area, the outer table of the skull remaining intact. A calcified pineal gland was shifted 10 mm. to the right of midline.

Left carotid angiography was performed on September 23, 1965. Frontal views revealed displacement of the anterior cerebral artery and internal cerebral vein to the right of midline, and the surface vessels did not reach the inner table of the skull in the area of thinning (Fig. 2). The sylvian vessels were displaced medially. Lateral views were normal except for slight straightening in the second portion of the anterior cerebral artery. The angiographic findings

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indicated a large, avascular, extracerebral mass overlying the left fronto-parietal convexity and extending into the temporal fossa. The mass failed to reach the vertex and lacked the characteristically biconvex contour of a chronic subdural hematoma. Clinical and roentgenographic findings led to a provisional diagnosis of cerebral leptomeningeal cyst.

The patient underwent craniotomy on September 27, 1965. The dura bulged outward over a broad area, ovoid in contour and with a clearly demarcated border conforming to the thinned area in the overlying skull. Reflection of the dura disclosed an arachnoid-like membrane through which aspiration produced clear, colorless fluid resembling cerebrospinal fluid. A fine, opaque white line marked the attachment of the membrane to the normal arachnoid over the convex surface of the frontal and parietal lobes. The surface of the brain between the medial border of the cyst and midline had a normal appearance and measured 4 cm. at the frontal pole and 8 cm. at the coronal suture (Fig. 3). Where the cyst was covered by bony calvarium, cyst wall lay in apposition to the inner dural surface.

Excision of the superficial portion of the membranous cyst wall opened a cavity containing 150–175 cc. of clear fluid. The fluid was aspirated and thereafter the cavity remained

dry. The cortical surface of the frontal, parietal and temporal lobes formed the concave medial surface of the cavity. The gyri and sulci had normal proportions and the brain did not appear to be compressed. The smooth, inner surface of the cyst was separated from the cerebral cortex by a layer of cerebrospinal fluid within the subarachnoid space. The cyst wall lying against the brain had the appearance of normal arachnoid and, when incised, proved to be the only structure separating cyst fluid from cerebrospinal fluid.

The cranial base formed the floor of the cavity with the membranous cyst wall lying in direct apposition to, but separating easily from, the dura. The exposed portion of the anterior cranial fossa extended medially to the cribriform plate where the cyst wall became inseparable from arachnoid covering the inferior frontal gyrus. The exposed portion of the middle fossa extended caudally onto the tentorium. The free edge of the tentorium and the ambient cistern were just visible beneath the inferior temporal gyrus (Fig. 3).

The cavity was filled with saline followed by routine craniotomy closure. The patient's course following surgery was smooth, and she left the hospital on the fourth postoperative day.

The excised portion of cyst wall had a histo-



FIG. 2. Frontal view of left carotid arteriogram showing displacement of anterior and middle cerebral arteries, and depression of cortical branches away from the calvarium.

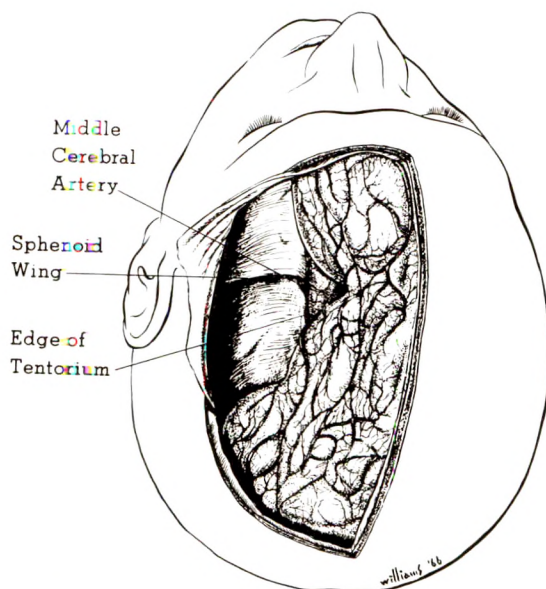


FIG. 3. Artist's drawing of the cyst exposed at the time of operation.



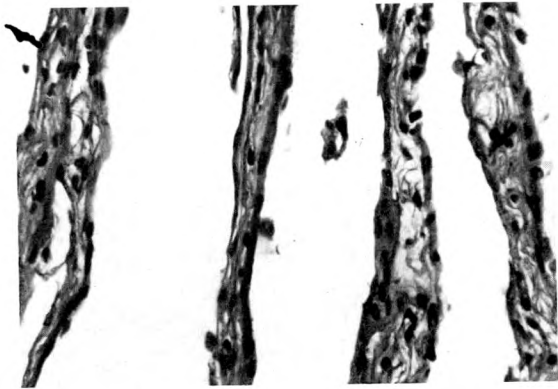


FIG. 4. Histologic appearance of the cyst wall.



FIG. 5. Frontal view of left carotid arteriogram made 6 weeks following operation.

logic appearance consistent with thickened arachnoid (Fig. 4). Mesothelial cells covered both surfaces of a thin membrane composed of collagen fibers. Evidence of inflammation was lacking. Fluid within the cyst contained 17 mg. per cent protein, 60 mg. per cent glucose and 2,340 red blood cells.

Six weeks following surgery, the patient entered the hospital for follow-up study. A left carotid angiogram showed no change from the preoperative angiogram (Fig. 5). A pneumoencephalogram revealed slight asymmetry of the ventricular system, and minimal deformity of the left lateral ventricle with displacement to the right side (Fig. 6). No air entered the cyst initially, or 24 hours later.

Six months following surgery, the patient denied symptoms. Weakness and sensory loss

had disappeared. Two headaches during the previous month were unlike her former headaches. Plain skull roentgenograms showed displacement of the pineal gland 3.5 mm. to the right of midline.

#### DISCUSSION

Starkman, Brown and Linell<sup>22</sup> published postmortem findings in a case almost identical to ours. Their report contains clinical information, roentgenologic studies, and gross and microscopic descriptions of the cyst and underlying brain. They demonstrated the position of the cyst between two layers of arachnoid. The critical evidence in their case was a microscopic description of the brain where cyst wall attached to the normal arachnoid. At this point the arachnoid split, the outer portion continuing as the free wall of the cyst and the inner portion maintaining the position and appearance of arachnoid over the concave surface beneath the cyst. Terming the cyst intra-arachnoid, they argued for its developmental origin. Robinson also presented convincing evidence for the congenital origin of leptomeningeal cysts involving the temporal fossa.<sup>18</sup>

Leptomeningeal cysts are known to follow trauma.<sup>9,11,17,20,23,24</sup> Taveras and



FIG. 6. Frontal view of pneumoencephalogram made 6 weeks following operation. No air enters the cyst.

Ransohoff<sup>23</sup> described the characteristic clinical and pathologic features of traumatic leptomeningeal cysts. Traumatic cysts develop beneath a fracture typically involving the parietal bone in a child and the diagnosis offers little difficulty. The etiologic role of trauma in less typical cases must be accepted with reservation.

Roentgenologic findings in the present case suggested the correct diagnosis before surgery. Localized thinning of the skull and the shift of midline structures indicated a long-standing mass. Angiographic demonstration of an avascular, extracerebral structure narrowed the diagnostic possibilities and, lacking other evidence of a neoplasm, favored a localized collection of fluid overlying the brain.

Air studies in previously reported cases have been diagnostic only in instances where air reached the cyst.<sup>17,22,25</sup> The cyst in our case did not fill with air following operation, even after 24 hours, and it seems unlikely that air would have entered the cyst before operation.<sup>22</sup> Air studies will not establish the diagnosis of an intra-arachnoid cerebral cyst unless air is injected directly into the cyst. Angiography should suggest the correct diagnosis, but verification must await gross visualization of the cyst. Displacement of the pineal gland, shown in the present case and in others,<sup>18-22</sup> does not necessarily indicate increased intracranial pressure which may, or may not, be associated with these cysts.

Operative findings in our case support the contention of Starkman and co-workers<sup>22</sup> that one variety of leptomeningeal cyst lies within the arachnoid, *i.e.*, intra-arachnoid. Cyst fluid in our case resembled cerebrospinal fluid, although no communication between the cyst and the subarachnoid space was visualized at the time of surgery or demonstrated by pneumoencephalography. We assume that the membranous cyst wall was permeable.

Localized thinning of the skull rarely occurs beyond childhood.<sup>3</sup> Accepted as an indication of a long-standing process, bone erosion produced by an underlying cyst,

with or without a generalized increase in intracranial pressure, can be explained by the water-hammer effect of cerebral pulsations.

Surgical management of leptomeningeal cysts has varied. Internal drainage into the subarachnoid space or cerebral ventricle is appealing in principle but lacks justification by proof of permanency. Simple removal of a superficial portion of the cyst wall has proved successful on short-term analysis in our case and in others. Return of the displaced pineal gland toward midline in the present case indicates an alteration in intracranial hydrodynamics. Almost certainly, the cyst wall is capable of regeneration, and only long-term follow-up will determine the effectiveness of simple drainage in the treatment of cerebral intra-arachnoid cysts.

#### SUMMARY

The pathogenesis of nontraumatic cerebral leptomeningeal cysts has seldom been documented. Clinical, roentgenologic and anatomic observations in a single case are presented. The cyst did not communicate with the subarachnoid space, and evidence favored an intra-arachnoid cyst of developmental origin. Clinical and roentgenologic features of this variety of leptomeningeal cyst should permit preoperative recognition.

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## RADIOLOGIC DETECTION OF ASTROCYTOMA INVOLVING THE CORPUS CALLOSUM\*

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THE corpus callosum is the main commissure connecting the cerebral hemispheres. In the midline sagittal section of the brain, it is an arched structure, about 10.0 cm. in length, lying at the bottom of the longitudinal fissure. Its anterior end, the genu, lies 4.0 cm. from the anterior pole and its posterior end, the splenium, which is the thickest part of the corpus callosum, lies 6.0 cm. from the occipital pole. Its upper surface is covered by a thin layer of gray matter, the indusium griseum. It forms the roof of the bodies of the lateral ventricles. The pericallosal arteries sweep around its anterior end in close proximity to the genu and run posteriorly on its upper surface.

The difficulty of establishing a clinical diagnosis of tumor of the corpus callosum is well known.<sup>1,5,7</sup> It is not surprising, therefore, that extension of an infiltrating malignant tumor of the brain into the corpus callosum is also difficult to detect on the basis of clinical findings. That this is a very frequent occurrence in malignant glioma is indicated by the work of Maxwell<sup>6</sup> in which 75 per cent of their small series of glioblastomas involved the corpus callosum, and by Bull and Rovit<sup>2</sup> who found approximately 25 per cent of their cases showing bilateral involvement of the brain, implying involvement of the corpus callosum. A study of 200 consecutive cases of glioblastoma performed at the National Hospital, Queen Square, by Professor Blackwood showed approximately 35 per cent involvement of the corpus callosum. In view of the frequency of corpus callosum involvement as demonstrated at autopsy, the rarity of establishing the diagnosis on the basis of air encephalography indicates clearly that the problem is not solved by this method. Dyke and Davidoff<sup>3</sup> reported only 8 cases

in which corpus callosum tumor was detected from a series of 3,000 air studies.

This paper deals with the radiologic findings in 72 cases of malignant astrocytoma affecting the corpus callosum, all of which have been subjected to postmortem examination. Radiologic examinations dealt with in this study are: angiography, air encephalography or ventriculography and gamma scanning. There were 46 males and 26 females in this series. Figure 1 shows the age distribution.

The cases were selected from the autopsy records of the National Hospital for Nervous Diseases. The roentgenograms, scans and clinical records were then obtained. All cases were reviewed by the authors twice, first by one of them separately, and then by both together at which time a synthesis of any differences of interpretation was made. Angiograms were reviewed first, when

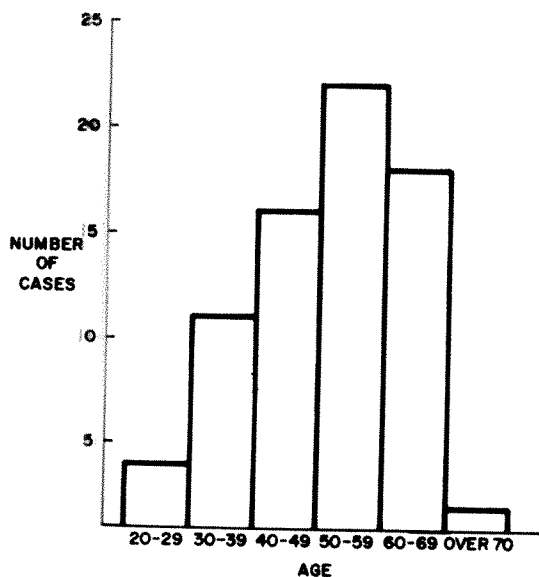


FIG. 1. Age distribution of patients with pathologically proven astrocytoma of the corpus callosum.

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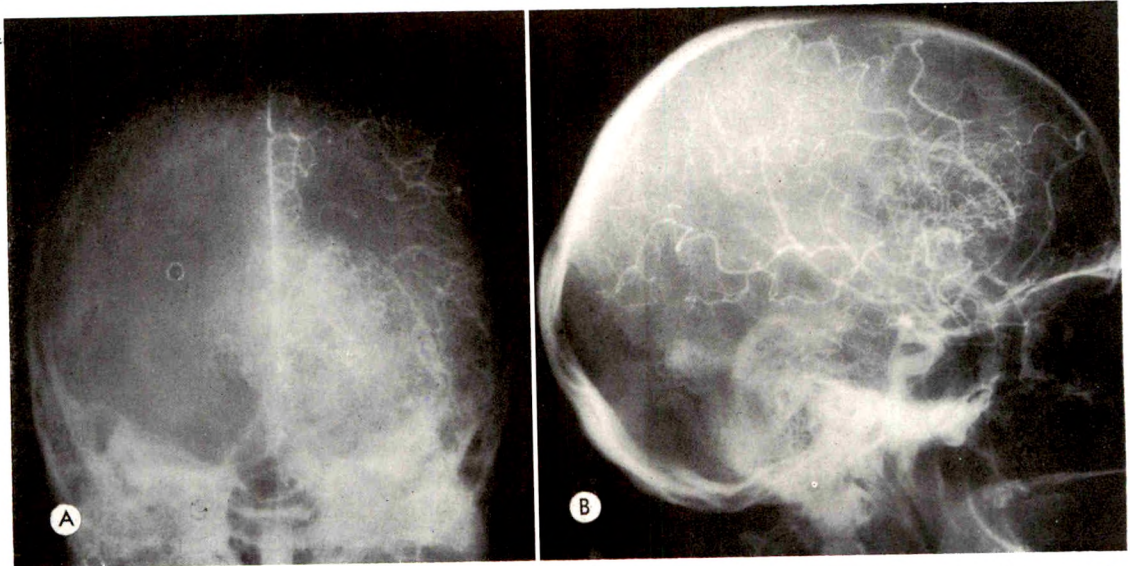


FIG. 2. (A) Anteroposterior and (B) lateral angiograms show pathologic vessels crossing the midline in the absence of displacement of the midline structures.

available, followed by air studies, gamma scans, clinical records and autopsy findings in that sequence. The criteria of involvement of the corpus callosum are as follows. First, on angiograms the exact location of



FIG. 3. Lateral venogram shows localized depression of the posterior part of the internal cerebral vein and widening of the sweep of the vein of Galen. Note that the curve of the vein is not flattened as with dilatation of the lateral ventricles.

the tumor can sometimes be seen due to the presence of pathologic vessels within the tumor itself. Often the extent of the tumor is greater than is shown by the pathologic circulation, but one may be sure that the abnormal vessels define the minimum extent of the tumor. Therefore, when the pathologic vessels are seen to extend across the midline in the region of the corpus callosum and there is not sufficient shift of the midline vessels to indicate that this appearance is due to herniation of the tumor mass under the falx, one may be certain that the tumor is infiltrating the corpus callosum (Fig. 2, A and B). Second, where there is localized depression of the posterior part of the internal cerebral vein and a widened sweep of the vein of Galen and callosal vein around the splenium of the corpus callosum, one may strongly suspect involvement of the splenium (Fig. 3), but, if the entire internal cerebral vein is depressed and flattened, the appearance can be due to enlargement of the lateral ventricles, so that one must be careful in his evaluation of this finding. Of the 35 angiograms, only 4 showed diagnostic findings, indicating approximately 11 per cent accuracy.



On the air studies, the most useful appearance has been widening of the septum pellucidum, associated with infiltration of the body of the corpus callosum (Fig. 4).

This appearance must be differentiated from cyst of the septum pellucidum and tumor of the septum pellucidum,<sup>4</sup> but in our experience other findings of a hemispheric mass usually make this differentiation quite simple. Deformity of the medial aspect of both frontal horns or of the medial aspect of the atria of both ventricles is not a specific finding of corpus callosum involvement, since this appearance may be produced by meningioma of the anterior or posterior part of the falx. Several of our cases have shown a well localized indentation of the roof of the bodies of the lateral ventricles which we thought must indicate corpus callosum involvement. This appearance is even more convincing when a distinct lobulation of the filling defect is visible. Naturally, the more localized the deformity of the ventricles, the more prone one is to ascribe it to tumor in the corpus callosum rather than lying directly above it. Twenty-five per cent (14 cases of 55) of the air studies were diagnostic of corpus callosum involvement and an additional nine per cent (5 cases) were regarded as suspicious.

Interpretation of the scans has presented some of the same problems as encountered

with contrast examination in that the presence of a zone of increased uptake of isotope crossing the midline could not, in itself, be considered evidence of interhemispheric extension in those cases where herniation of the tumor mass could produce this appearance. The most convincing appearance on the gamma scan is the "dumbbell"-shaped zone of increased uptake produced by a tumor mass in each hemisphere and its extension through the corpus callosum (Fig. 5). Of 6 cases in this series which had gamma scans, 5 (83 per cent) were positive.

Because the series of autopsy-confirmed scans was so small, it was elected to review the available scans of biopsy-proven astrocytoma cases in order to determine the percentage in which the scan gave evidence of corpus callosum involvement. Comparison of this percentage with the percentage of astrocytomas involving the corpus callosum at autopsy should give some insight into the sensitivity of the procedure applied to this specific problem. This result does not have the validity of a series of autopsy-proven cases, but will serve as a preliminary estimate until a more definite result can be obtained. Seventy-nine biopsy-proven astrocytomas on which scans had been performed yielded 14 cases in which evidence of corpus callosum involvement was regarded as conclusive (17.9 per cent) and 4 cases in which findings were thought to be



FIG. 4. Marked widening of the septum pellucidum and separation of the bodies of the lateral ventricles.



FIG. 5. "Butterfly" distribution of increased uptake of isotope. This is the most characteristic pattern of midline transgression.



suspicious (5 per cent). If one assumes on the basis of the autopsy evidence that approximately 35 per cent of this series would show corpus callosum involvement, there should be 28 such cases out of the 79 astrocytomas. This suggests 64 per cent accuracy for the gamma scans.

#### DISCUSSION

The fact that astrocytomas often grow by infiltrating normal brain tissue rather than by displacing it may account for the relatively low diagnostic accuracy of air studies and angiography.

A limitation of this study was that the method of selection of cases introduced the possibility of bias in interpretation of the roentgenograms since the authors were aware that corpus callosum involvement had been found at postmortem examination. For this reason, care was exercised to regard as positive only those cases in which masses in other locations would not be expected to produce a similar picture. There were many cases in which suspicion of corpus callosum involvement would have been amply justified, especially in view of the knowledge that such involvement is quite common, but these were not considered diagnostic of corpus callosum involvement when deformity might have been produced by tumor adjacent to the corpus callosum or secondary to herniation of the brain. Furthermore, one might rationally suspect that any method which deals with these studies as separate entities introduces a limitation beyond that which is experienced in ordinary usage where they can be combined to yield a maximum of information. For example, the combination of a contrast study showing the position of anatomic landmarks with a scan showing the extent of a tumor should allow much

more accurate localization than either study alone.

It should also be noted that the estimated 35 per cent of the non-proven cases in which corpus callosum involvement was assumed to be present is probably high, since this percentage was derived from autopsy figures. Some of the cases might have shown corpus callosum involvement had the scans been performed immediately prior to death, by which time some of the tumors might have infiltrated across the midline.

#### SUMMARY

Comparison of the accuracy of air studies, angiography and gamma encephalography in detection of involvement of the corpus callosum by malignant astrocytoma reveals a clear superiority of the gamma scanning technique.

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## ANGIOGRAPHIC FINDINGS IN AGENESIS OF THE CORPUS CALLOSUM

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THE corpus callosum originates from the lamina terminalis in the third and fourth month of fetal life. Before its formation, radially arranged furrows can be seen on the medial aspect of the two hemispheres. Normally, these are transitory structures, but they may persist wherever the corpus callosum fails to develop. This is particularly well shown in patients with partial agenesis in that the furrows persist where the corpus callosum has not developed but are not seen in those areas where it is present. These conditions are significant for the angiographic diagnosis of agenesis of the corpus callosum.

Agenesis of the corpus callosum may be classified among the dysraphias and may be partial or total. The causal factors have not been clarified. Disturbances of the fetal circulation in the anterior cerebral arteries have been suggested and hereditary factors

may have some significance. Zellweger<sup>7</sup> reported agenesis of the corpus callosum in 2 brothers.

The anomaly frequently occurs in combination with other malformations such as hydrocephalus, porencephaly and inter-hemispheric cysts. Formation of a lipoma in association with corpus callosum may also occur. It is maintained that the clinical symptoms are not due to the agenesis but the accompanying cerebral lesions. Oligophrenia is usually seen, but otherwise the symptomatology is varied and asymptomatic cases have been described.

Agenesis of the corpus callosum cannot be diagnosed clinically. Until now, the diagnosis has generally been made with the help of pneumoencephalography, but it can also be made with carotid angiography.<sup>1-3, 5-8</sup> A case diagnosed angiographically is presented below.

### REPORT OF A CASE

The patient was a 24 year old seaman admitted to the neurological clinic of the Medical School of the University of Bergen (Chief: Arne Galtung Frøvig) with the diagnosis of traumatic encephalopathy. He had had three head injuries; with one, there was a possible loss of consciousness. He was later troubled with headache, reduced memory, diminished concentration, nervousness and irritability. A routine neurologic examination showed no pathologic findings apart from moderate mental deficiency. Electroencephalography under standard conditions showed a generalized cerebral dysrhythmia with an excess of beta activity.

Roentgenograms of the cranium showed no abnormal findings. A pneumoencephalogram (Fig. 1), however, revealed considerable separation of the lateral ventricles, particularly of the body and anterior horn, as well as a distinctly enlarged temporal horn. From these findings, the possibility of agenesis of the corpus callosum or a noncommunicating cyst in the septum pellucidum was considered.

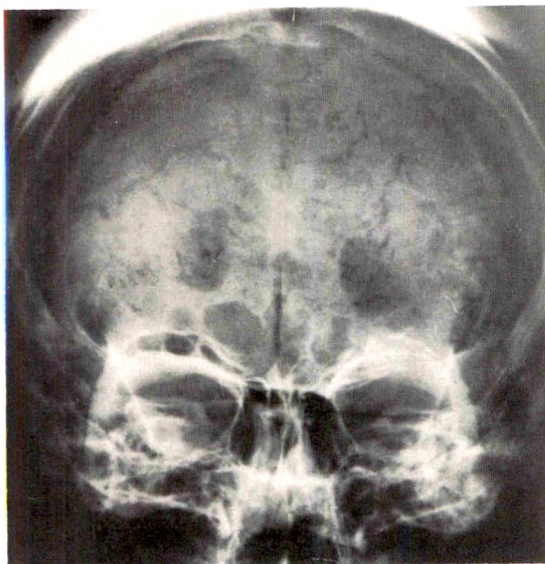


FIG. 1. Anteroposterior pneumoencephalogram shows poor filling of the ventricles, which are widely separated. There is no definite filling of the third ventricle.



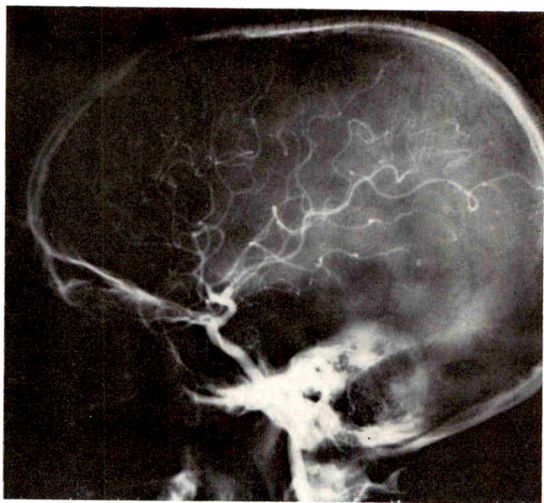


FIG. 2. Arterial phase of right sided lateral projection. In this study it is difficult to identify the pericallosal artery.

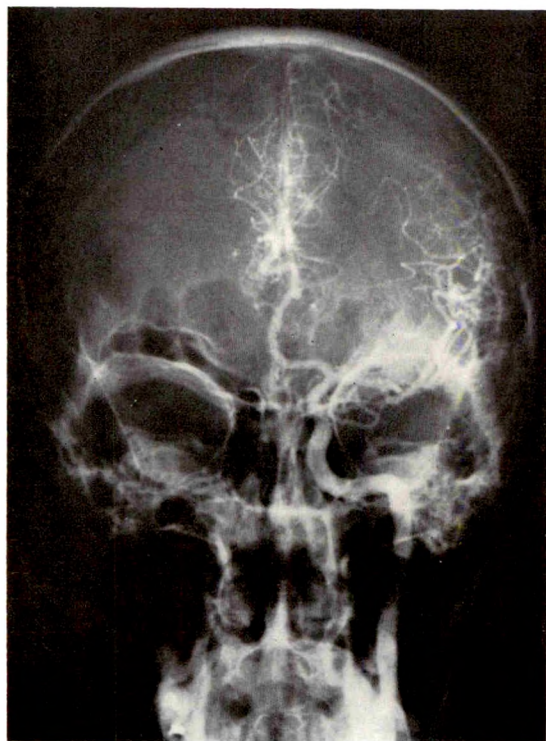


FIG. 3. Arterial phase of left sided anteroposterior projection. The wavy character of anterior cerebral and pericallosal arteries is seen.

Air encephalography was not very successful technically and was difficult to assess. Bilateral carotid angiography was, therefore, carried out (Fig. 2 through 5). The angiographic findings,

some of which do not appear to have been described before, were interpreted as typical for agenesis of the corpus callosum. The anterior cerebral arteries were asymmetric, the right being of small caliber and the left considerably larger. Both lacked the normal arched course around the genu of the corpus callosum seen in lateral projection. The left anterior cerebral artery rose steeply in an anteriorly concave arch. At the level of the origin of the callosomarginal artery, it curved sharply backwards and divided immediately into two branches of approximately equal thickness. Three larger



FIG. 4. Arterial phase of left sided lateral projection. (See text for details.)

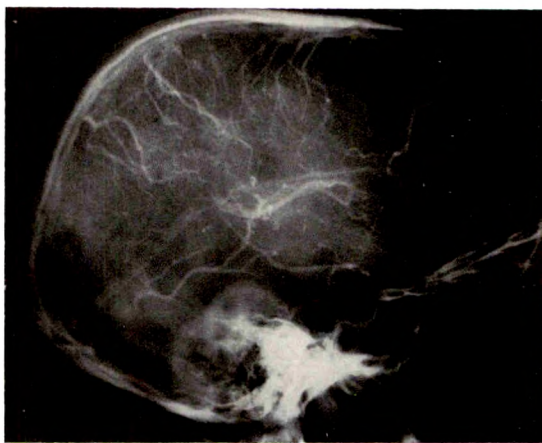


FIG. 5. Venous phase of left sided lateral projection. Note the close relationship between the internal cerebral vein and the inferior sagittal sinus. The diminished curvature of the great cerebral vein is seen. The thalamostriate vein lies below the internal cerebral vein. The septal vein was more prominent on later angiograms in the series.

branches of the callosomarginal artery ran a distinctly radiating course towards the vertex and it is possible that they coursed into the aforementioned furrows. The medial cerebral arteries showed normal relationships on both sides.

The relations of the veins were interesting. The internal cerebral vein and the great cerebral vein (great vein of Galen) lay higher than normal and, at the same time, the venous angle lay too far cranially and ventrally. This was probably due to enlargement of the third ventricle in a cranial and ventral direction.

In contrast to previous reports,<sup>1,3</sup> a normally developed vein of the septum pellucidum was found on both sides. However, in order to reach the internal cerebral vein, this vein rose higher than is usual as it curved backwards. The thalamostriate vein showed an even more unusual course. From its position in the lateral ventricle, it had to run *upwards* and medially on both sides in order to empty into the internal cerebral vein. The smaller branches of the thalamostriate vein extended far from the midline. This could only be explained on the basis of the separation of the lateral ventricles as seen with agenesis of the corpus callosum. A similar finding occurs with internal hydrocephalus, but in that condition, the great cerebral vein lies lower than normal rather than higher, as in this case.

After receiving the inferior sagittal sinus, the great cerebral vein did not run directly into the straight sinus. Instead, there was a relatively small-calibered connection between these 2 structures. A similar variant was demonstrated by Claus and Heidrich<sup>1</sup> in their patient with agenesis of the corpus callosum.

#### DISCUSSION

Angiographically, the diagnosis of agenesis of the corpus callosum can be based on the following findings (Fig. 6):

1. The reduced distance between the inferior sagittal sinus and the internal cerebral vein.
2. The reduced distance between the pericallosal artery and the internal cerebral vein.

Both of these findings depend on the presence of an abnormally reduced distance between structures which lie above and below the corpus callosum. Thus, a direct

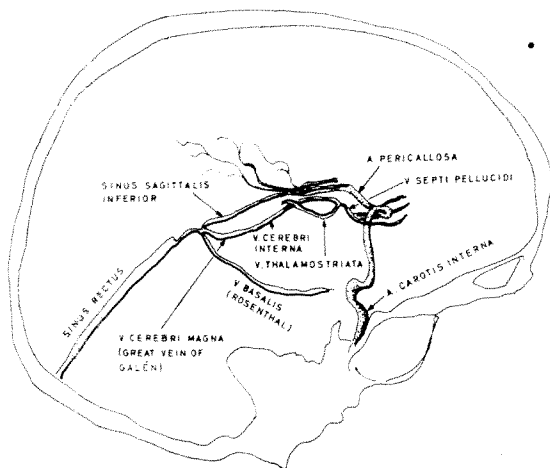


Fig. 6. Sketch of the deep veins. The internal carotid artery, the anterior cerebral artery and the pericallosal artery are added to show the relationship between these arteries and the internal cerebral vein. Between the internal cerebral vein and the pericallosal artery, there is no room for a normal corpus callosum.

angiographic diagnosis of agenesis of the corpus callosum is possible by comparing the course of the vessels on the upper side of the corpus callosum with the course of those on the under side.

Morris<sup>5</sup> suggests that in agenesis of the corpus callosum there is a separation of the two internal cerebral veins. This was not found by Zingesser *et al.*<sup>8</sup> The relation seems to be dependent on the projections used. In the enlarged third ventricle, the internal cerebral vein lies laterally at its origin at the foramen of Monro but it approaches the median plane before joining the vein from the other side to form the great cerebral vein, as in the case reported here.

The arteries of the sylvian group were localized higher than normal in 1 of the patients reported by Zellweger,<sup>7</sup> in 1 of Holman and MacCarty's 2 patients,<sup>3</sup> and in the 1 case reported by Claus and Heidrich.<sup>1</sup> However, as a rule, these arteries run a normal course, as in our patient.

#### SUMMARY

A patient with agenesis of the corpus callosum is reported. Angiographic demon-



stration of a reduced distance between the inferior sagittal sinus and the internal cerebral vein, and between the pericallosal artery and the internal cerebral vein is assumed to be evidence of agenesis of the corpus callosum.

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## THE ROENTGENOLOGY OF THE MENINGO-HYPOPHYSEAL TRUNK\*

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THE internal carotid artery within the cavernous sinus supplies the hypophysis, the optic chiasm, the semilunar ganglion and, in addition, gives off several branches to supply the tentorium. Many of these anastomose with similar branches from the contralateral internal carotid artery while others anastomose with the middle meningeal artery (Fig. 1; and 6, *A* and *B*).<sup>9,11</sup>

### THE BLOOD SUPPLY OF THE HYPOPHYSIS

The arterial blood supply of the hypophysis is derived from the inferior and superior hypophyseal arteries which arise from the carotid siphon. The inferior hypophyseal artery supplies the posterior lobe while the superior hypophyseal artery supplies the anterior lobe.<sup>9</sup> The inferior hypophyseal artery arises from a common trunk which takes origin at the junction of the proximal vertical and horizontal segments of the cavernous internal carotid artery. The trunk from which it arises may be termed the meningo-hypophyseal trunk<sup>11</sup> since it distributes 3 branches which supply the hypophysis and the meninges. The branches are respectively: the inferior hypophyseal artery, the dorsal meningeal artery, and the tentorial artery (Fig. 2 through 8). The inferior hypophyseal artery passes upwards and medially to reach the lateral surface of the posterior lobe of the hypophysis where it divides into superior and inferior divisions and anastomoses with similar branches from the other side. The dorsal meningeal artery passes behind the dorsum sellae to ramify on the posterior surface of the clivus, and anastomose with similar branches from the contralateral internal carotid artery. The tentorial artery

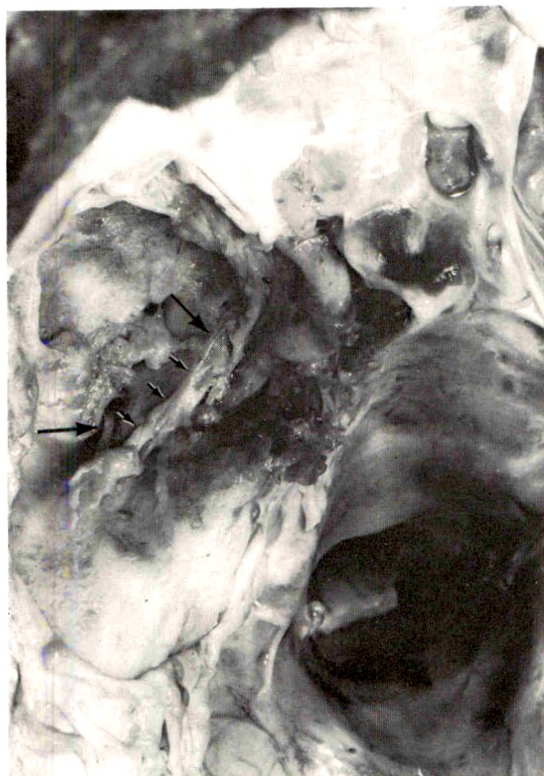


FIG. 1. Dissection of the cavernous portion of the internal carotid artery, showing artery to the inferior cavernous sinus (upper large arrow and small arrows) and its anastomosis with the middle meningeal artery (lower large arrow). The artery has not been entirely freed from the dura.

passes backwards within the edge of the tentorium. It also anastomoses with its fellow from the opposite internal carotid artery.

In many instances, another trunk arises from the lateral aspect of the cavernous internal carotid artery which supplies the structures of the inferior cavernous sinus. This vessel, which has been termed the artery to the inferior cavernous sinus, anasto-

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FIG. 2. Dissection of branches of the cavernous portion of the internal carotid artery. The tentorial artery (left arrow) arises from the junction of the proximal vertical and horizontal segments of the internal carotid artery within the cavernous sinus. On the right (middle arrow) a common trunk is noted which divides into two. An anterior branch (the inferior hypophyseal artery) runs forwards to supply the hypophysis while the posterior branch (the dorsal meningeal) passes behind the dorsum to supply the meninges. The trigeminal nerve (right arrow) is seen parallel to the internal carotid artery.

moses directly with the middle meningeal artery and thus is instantly available as a collateral channel in cases of carotid occlusion, carotid cavernous fistula, and arteriovenous malformations (Fig. 1; and 6, *A* and *B*).

McConnell's<sup>9</sup> description of the blood supply of the hypophysis differs slightly from the above, but was considered by her to be remarkably constant with only occasional minor variations. She described the trunk as arising from the medial aspect at about the center of the cavernous internal carotid artery, and also describes 3 branches: an anterior running downwards and laterally to the cavernous sinus, a posterior running backwards and medially behind the dorsum sellae, and the main trunk itself which continues as the inferior hypophyseal artery. It was our impression from dissections of the cavernous portion of the internal carotid artery that these arteries are not as constant as previously described, and that there is considerable variation in the origin of these vessels. Thus, while one may be able to demonstrate a classic meningo-

hypophyseal trunk on one side, the various branches may arise individually on the opposite side (Fig. 2; and 3). We feel that McConnell<sup>9</sup> and Parkinson<sup>11</sup> are probably describing the same vessels but with different terminology, depending on the origin of these vessels.

The superior hypophyseal arteries arise from the posteromedial aspect of the internal carotid artery shortly after it emerges from the cavernous sinus, this being the intradural supracavernous portion of the internal carotid artery. The arteries run upwards and medially to supply the optic nerve, the optic chiasm, and the anterior lobe of the hypophysis (Fig. 4, *A*, *B* and *C*).

The meningo-hypophyseal arteries are seldom seen in a normal arteriogram, but may be found enlarged in pathologic conditions.<sup>1-3,5,12,13,15,16</sup> Thus, we have identified these vessels in 2 pituitary adenomas, a hypothalamic glioma, and a falx meningioma. In addition, the anastomosis between the artery to the inferior cavernous sinus and the middle meningeal artery was demonstrated in 1 case (Fig. 6, *A* and *B*). The tentorial artery has been identified by other authors<sup>1-3,5,12,13,15,16</sup> in the blood supply of the tentorial meningiomas, arteriovenous malformations involving the tentorium, gliomas invading the tentorium,

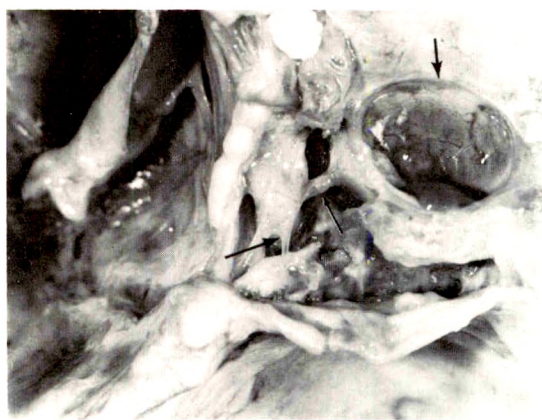


FIG. 3. Dissection of the cavernous sinus showing the pituitary (right upper arrow), the meningo-hypophyseal trunk (middle arrow) at its origin from the cavernous internal carotid artery, and the tentorial branch (left lower arrow).



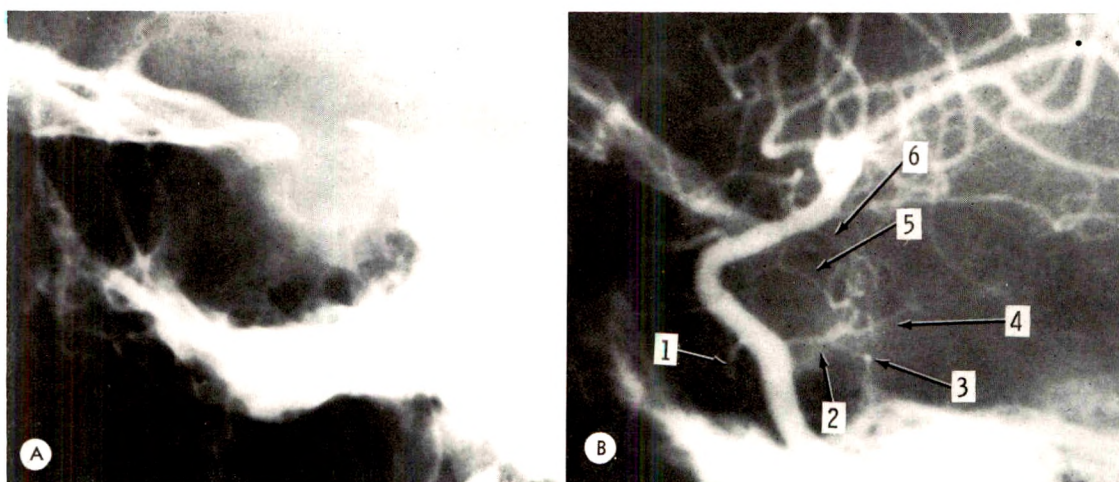


FIG. 4. (A) Plain roentgenogram of the skull in a case of chromophobe adenoma with parasellar and suprasellar extension. The sella turcica is ballooned, the lamina dura has been destroyed, and there is a double floor. A soft tissue mass overlies the sphenoid sinus. (B) Internal carotid arteriogram demonstrates: (1) artery to the inferior cavernous sinus; (2) the meningo-hypophyseal trunk; (3) the tentorial artery; (4) the dorsal meningeal artery; (5) the hypophyseal artery; and (6) the superior hypophyseal arteries. A cluster of abnormal vessels can be seen between the apex of arrow 4 and the shaft of arrow 5.

trigeminal neurinomas, and in a number of other conditions (Table 1). Our findings will be described in some detail.

#### THE BLOOD SUPPLY OF THE TENTORIUM

While McConnell's<sup>9</sup> paper was mainly concerned with the blood supply of the hypophysis, later investigators demonstrated that, in addition to the arteries she described, the cavernous portion of the internal carotid artery gives off additional branches, some of which supply the tentorium. Interest in these branches was generated by the report of Bernasconi and Cassinari<sup>1</sup> in 1956, who described a tentorial artery in cases of tentorial meningioma. Other reports followed, and for a while it was believed that the demonstration of such an arterial supply was pathognomonic of a tentorial meningioma. However, this did not prove to be correct. Schnürer and Stattin<sup>12</sup> injected the internal carotid artery and described in some detail the anatomy of the tentorial branches of the internal carotid artery.

Parkinson,<sup>11</sup> from the dissection of 200 cadavers, describes the tentorial artery as a branch of a common trunk which arises

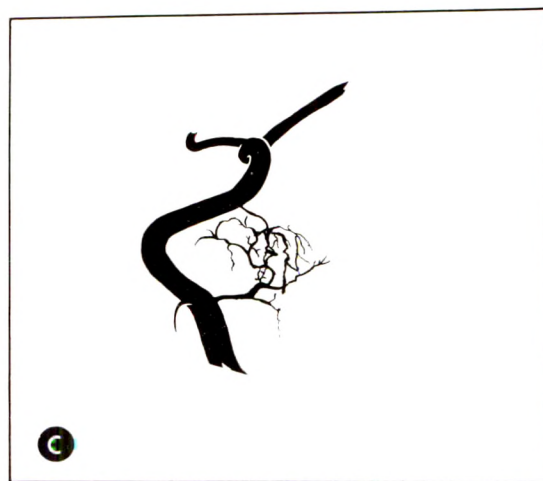


FIG. 4. (C) Sketch of B.

from the vertical segment of the cavernous portion of the internal carotid artery shortly after it enters the cavernous sinus. This artery, too, is seldom seen in the normal arteriogram, but it has been identified on a number of occasions in pathologic conditions. We have seen it in cases of pituitary and hypothalamic tumor, an extradural mass, a falx meningioma, and a scalp angioma (Fig. 4 through 8).

In addition, we have found that the ten-



TABLE I

Authors	Meningioma	Arteriovenous Malformations involving Tentorium	Gliomas invading Tentorium	Trigeminal Neurinoma	Scalp Angi- oma	Pituitary Tumor	Hypothal- amic Tumor	Other
Bernasconi and Cassinari (1956) <sup>1</sup>	5 tentorial	—	—	—	—	—	—	—
Krayenbühl and Yasargil (1957) <sup>6</sup>	—	1	—	—	—	—	—	—
Wickbom and Stattin (1958) <sup>16</sup>	2 tentorial	—	—	—	—	—	—	—
Frugoni <i>et al.</i> (1960) <sup>3</sup>	8 tentorial	—	—	—	—	—	—	—
	2 falx	—	—	—	—	—	—	—
	1 parasagittal	—	—	—	—	—	—	—
Stattin (1961) <sup>13</sup>	8 tentorial	2	—	—	—	—	—	5
Westberg (1963) <sup>15</sup>	—	—	—	4	—	—	—	—
Cortes, Chase, and Leeds (1964) <sup>2</sup>	—	3	1	—	—	—	—	—
Kramer and Newton (1965) <sup>6</sup>	1 tentorial	1	1	1	—	—	—	—
Pribram (1966)	1 falx	—	—	—	1	2	1	1

torium receives branches from the anterior inferior cerebellar artery (Fig. 9). Branches of the extracranial vertebral artery, the occipital artery, as well as the posterior cerebral arteries, also supply the tentorium. We would anticipate that, with the increasing detail obtainable with subtraction and magnification methods, the other tentorial branches will be identified at angiography.

THE TRIGEMINAL ARTERY

The trigeminal artery arises from the cavernous internal carotid artery at the same point as the meningo-hypophyseal trunk, so that one may be mistaken for the other.<sup>6</sup> Since the significance of these vessels is somewhat different, it is pertinent to review the anatomy of the trigeminal artery.

In the 3 mm. embryo, the trigeminal artery connects the intracranial internal carotid arteries with the two vessels which later form the basilar artery. The trigeminal arteries are normally obliterated by the 14 mm. stage, but occasionally one may persist into adult life.<sup>10</sup> The persistent trigeminal artery has been described on a number of occasions<sup>8,14</sup> and we expect to demonstrate 1 in every 600 arteriograms. The trigeminal artery also arises from the medial aspect of the junction of the proximal vertical and horizontal segments of the cavernous internal carotid artery; it then passes medially and posteriorly either through the dorsum sellae or lateral to it to join the basilar artery (Fig. 10, A–I).<sup>8</sup> In

association with this artery may be found several other anomalies such as aneurysm, angioma, absence of the vertebral arteries, absence of the posterior communicating arteries, absence of the proximal basilar artery, and in 1 case we demonstrated an anomaly in the origin of the great vessels from the aorta. To date, no case of bilateral trigeminal arteries has been recorded.

In general, the trigeminal artery is larger than the meningo-hypophyseal trunk, although we have 1 example of a very small trigeminal artery (Fig. 10A). Since the trigeminal artery joins the basilar artery, a short vertical segment of the basilar artery can usually be identified, and often the posterior cerebral and superior cerebellar arteries also. The associated anomalies of the persistent trigeminal artery are vascular (angioma or aneurysm), whereas the meningo-hypophyseal trunk is more often associated with extradural or tentorial masses. However, it may also supply arteriovenous malformations and gliomas.

ANGIOGRAPHY

The visualization of the meningo-hypophyseal branches of the internal carotid artery, at angiography, is aided by selective angiography of the internal carotid artery, as superimposition of the branches of the external carotid artery, particularly the superficial temporal artery, otherwise makes identification incredibly difficult. Even with selective angiography, branches of the middle cerebral artery may be superim-

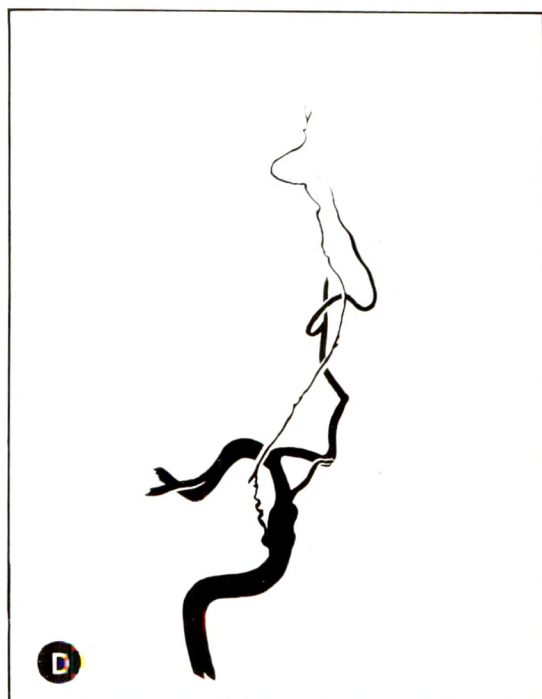
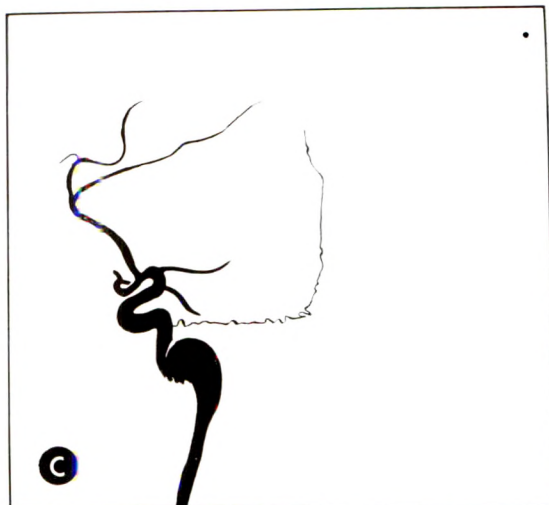
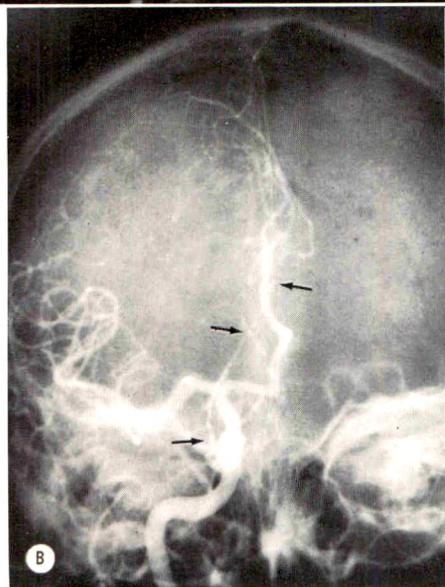
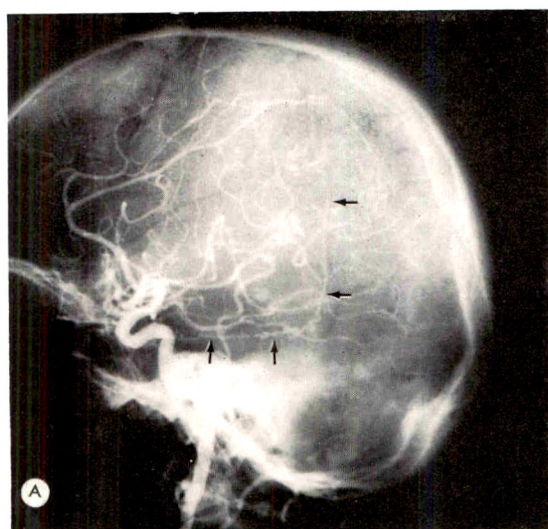


FIG. 5. Falx meningioma. (A) Lateral carotid arteriogram showing blood supply from tentorial artery (arrows). (B) Frontal arteriogram shows course of tentorial artery along the edge of the tentorium (arrows).

FIG. 5. (C) Sketch of A. (D) Sketch of B.

posed on the branches of the cavernous internal carotid artery. Stereoscopy in such instances may be justified.

#### REPORT OF CASES

CASE I. Chromophobe adenoma with suprasellar and parasellar extension. F.V., male, aged 33 years, with progressive loss of vision, blindness of the right eye, and severe loss of vision in

the left eye. Skull roentgenograms (Fig. 4A) showed an enlarged sella turcica with a soft tissue mass overlying the sphenoid sinus. The right superior orbital fissure was enlarged and the inferior margin of the right optic foramen was destroyed. Angiography revealed an intrasellar mass with suprasellar extension under the left frontal lobe, and evidence of bilateral extradural parasellar extension. The venous angle was displaced upwards and backwards, and the



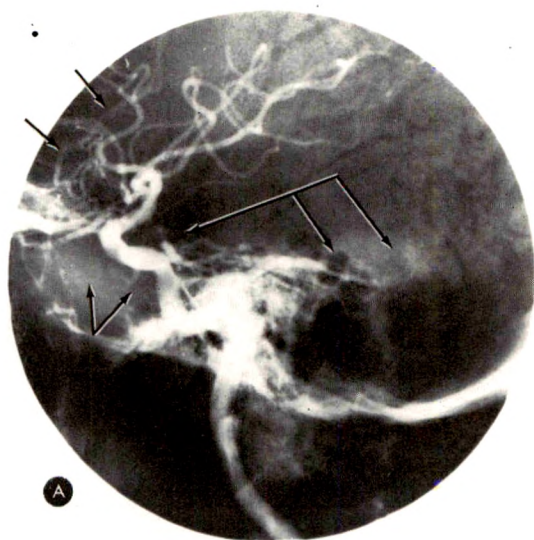


FIG. 6. Case of scalp angioma in which the external carotid artery has been ligated. (A) The anastomosis between the artery to the inferior cavernous sinus and the middle meningeal artery is seen (left arrows). A large meningo-hypophyseal trunk (right arrows) arises from the junction of the proximal vertical and horizontal segments of the extradural internal carotid artery. The branches run laterally in the tentorium to supply the scalp angioma. Increased arterial and venous markings can be seen in the parietal bone.

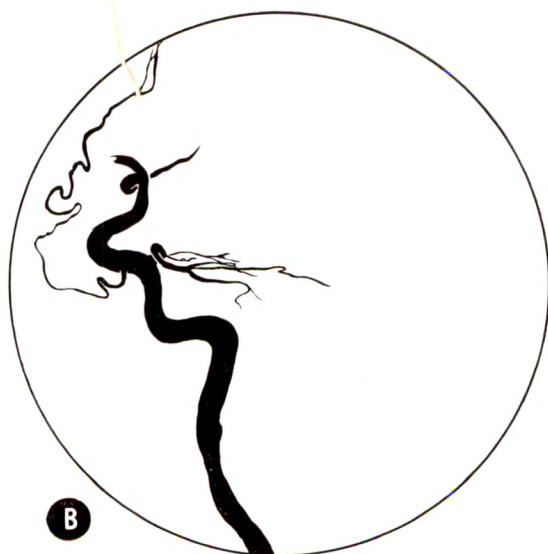


FIG. 6. (B) Sketch of A.

ventricles were enlarged. A left carotid arteriogram (Fig. 4, B and C) demonstrated the meningo-hypophyseal trunk and its branches, as well as the artery to the inferior cavernous sinus which was displaced downwards and laterally. A cluster of abnormal vessels derived from the inferior hypophyseal artery was noted. A right carotid arteriogram showed a small meningo-hypophyseal trunk and evidence of parasellar

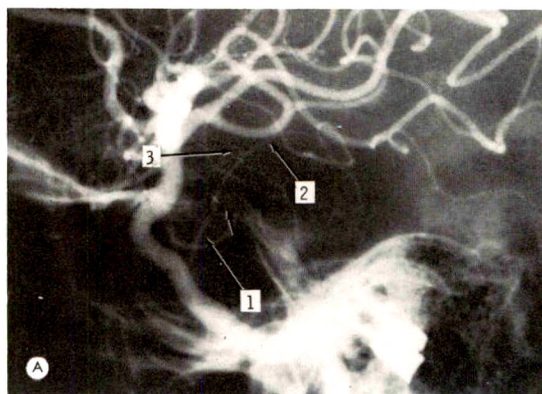


FIG. 7. Extradural mass. (A) The proximal vertical and horizontal segments of the extradural internal carotid artery are displaced forwards. The meningo-hypophyseal artery (1) is shown with its hypophyseal (3) and tentorial (2) branches. The dorsal meningeal artery is crossed by the small arrow.

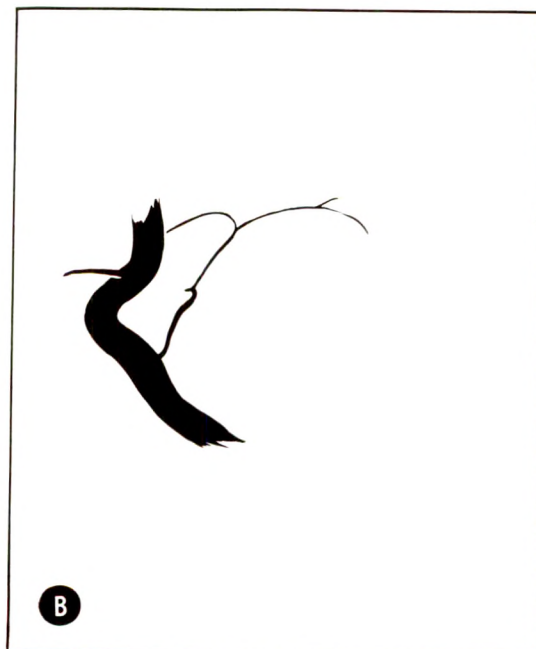


FIG. 7. (B) Sketch of A.



extension. Surgery revealed a chromophobe adenoma.

*Comment.* This was an extremely large tumor with large extradural middle fossa components which resulted in unveiling of the branches arising from the cavernous

portion of the internal carotid artery. Lateral and downward displacement of the artery to the inferior cavernous sinus placed the mass within it. This is the first occasion that we have ever demonstrated pathologic vessels in a chromophobe adenoma. The presence of abnormal vessels made us favor a diagnosis of pituitary adenoma rather than that of craniopharyngioma. Abnormal vessels would be more likely in large or rapidly growing adenomas. We are aware of only 1 case in the literature where a tumor stain has been reported.<sup>7</sup>

**CASE II.** Chromophobe adenoma with suprasellar extension. A.H., female, aged 63 years, with panhypopituitarism and a bitemporal hemianopia with considerable loss of vision in the left eye. Plain roentgenograms of the skull showed the sella turcica to be enlarged. Initially, she was treated with radiation therapy which resulted in some improvement in her visual acuity, but was followed by considerable loss of vision in the left eye. Angiography (Fig. 11) showed evidence of suprasellar extension of an intrasellar mass, particularly on the left side. The meningo-hypophyseal trunk was identified, but no abnormal vessels were noted. Surgery revealed a chromophobe adenoma.

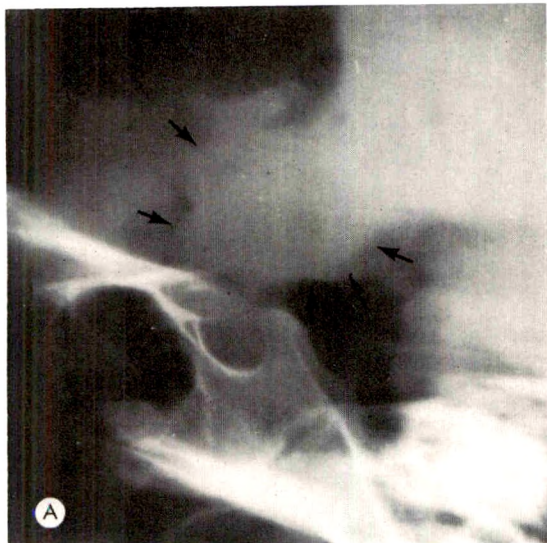


FIG. 8. Glioma of the hypophysis. (A) Air encephalogram showing hypothalamic mass (arrows) which partially occupies the interpeduncular cistern.

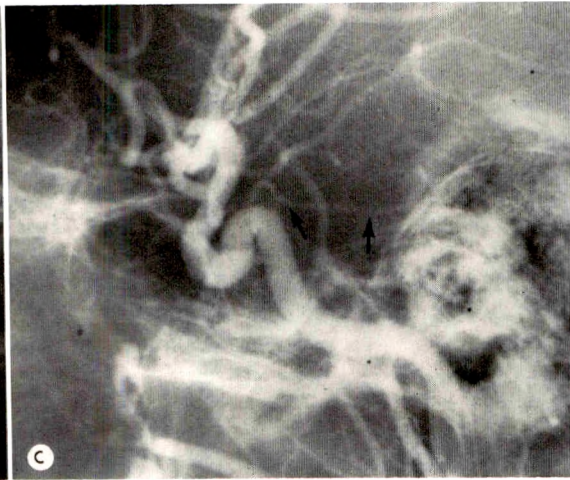
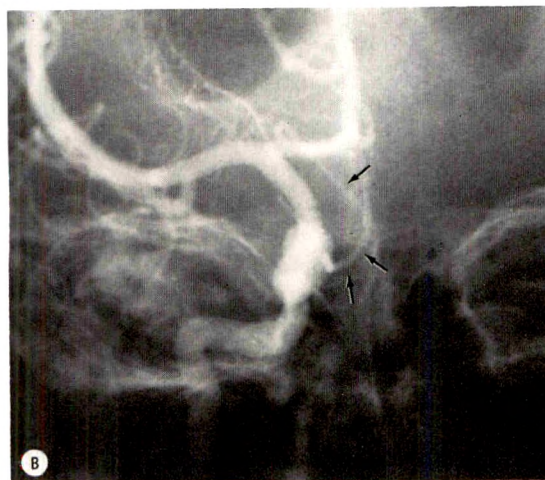


FIG. 8. (B) Frontal arteriogram showing an artery arising from the medial aspect of the cavernous internal carotid artery, coursing medially and then posteriorly (arrows). This is probably the hypophyseal artery rather than the tentorial artery. Compare the course of this artery with that of the tentorial artery in Figure 5B. (C) Lateral carotid arteriogram showing the hypophyseal artery running medially and upwards, then downwards and posteriorly in the interpeduncular cistern. The artery has been displaced downwards by the mass which occupies the upper part of the interpeduncular cistern (see Fig. 8A). (Courtesy Dr. J. D. R. Miller, University Hospital, Edmonton, Alberta, Canada.)



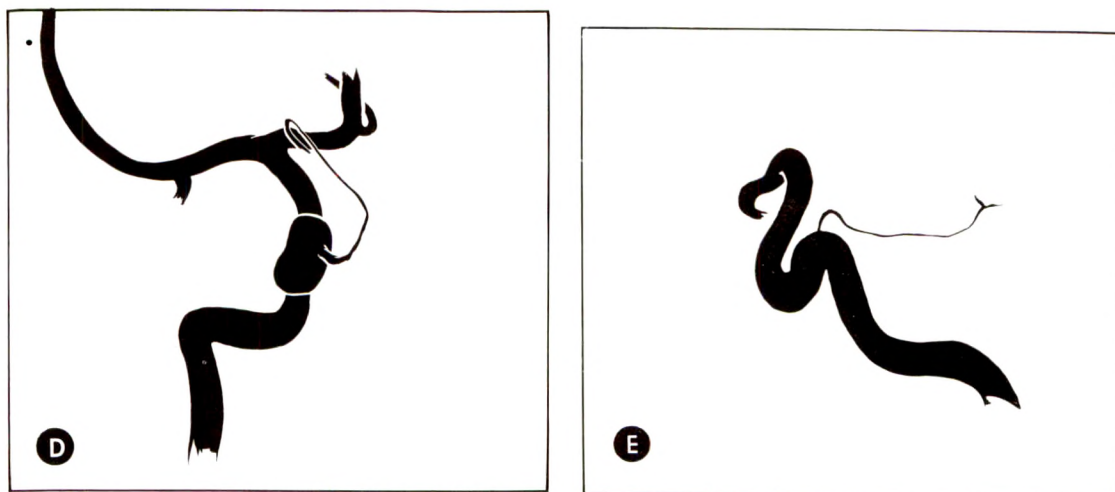


FIG. 8. (D) Sketch of B. (E) Sketch of C.

*Comment.* The identification of the meningo-hypophyseal trunk is probably a reflection of both the size of the tumor and the size of its extradural component.

CASE III. Scalp angioma. D.H., female, aged 21 years, with a right sided scalp angioma. Plain roentgenograms of the skull showed a considerable increase in the arterial and venous markings of the right side of the skull, and angiography revealed abnormal vessels in the right side of the scalp which derived their main blood supply from the external carotid artery. The right external carotid artery was then ligated. A year later she was re-admitted with complaints of loud intracranial bruits and head-

ache. Total angiography was performed. The most significant findings were at right carotid angiography which showed a large meningo-hypophyseal trunk supplying the scalp angioma (Fig. 6, A and B). The artery to the inferior cavernous sinus was also demonstrated. It was considerably enlarged, and its many small hypertrophied branches filled the middle meningeal artery which supplied the angioma.

*Comment.* The potential collateral pathway through the anastomosis between the artery to the inferior cavernous sinus and the middle meningeal artery has been appreciated only recently (Fig. 1). This is the first time that we have been able to demon-



FIG. 9. Dissection to show origin of tentorial branches (left horizontal arrows) from the anterior inferior cerebellar artery (left vertical arrow). The trigeminal nerve is seen (right horizontal arrow).

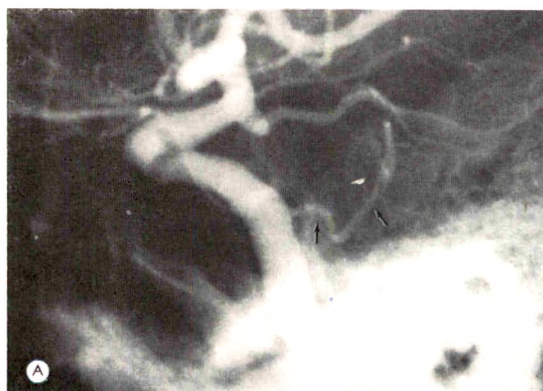


FIG. 10. Trigeminal artery. (A) Small trigeminal artery which might be mistaken for a tentorial artery.

strate this anastomosis at angiography. The pathway, of course, is one of the reasons that a carotid cavernous fistula may be difficult to obliterate even when the intracranial and extracranial internal carotid artery has been ligated. Furthermore, this pathway is a collateral route in cases of carotid occlusion.

CASE IV. Falx meningioma. M.H., female, aged 62 years, with complaints of headache, convulsions, and weakness of the left arm and leg. Right carotid angiograms (Fig. 5, A-D)

showed a large right sided falx meningioma, supplied in part by an enlarged tentorial artery which ran along the edge of the tentorium and then upwards along the falx to supply the meningioma. Surgery revealed a large falx meningioma.

*Comment.* The presence of a tentorial artery has been known to neurosurgeons who have had occasion to incise the tentorium. It has only recently been demonstrated at angiography, and is most often recognized in association with tentorial or falx menin-

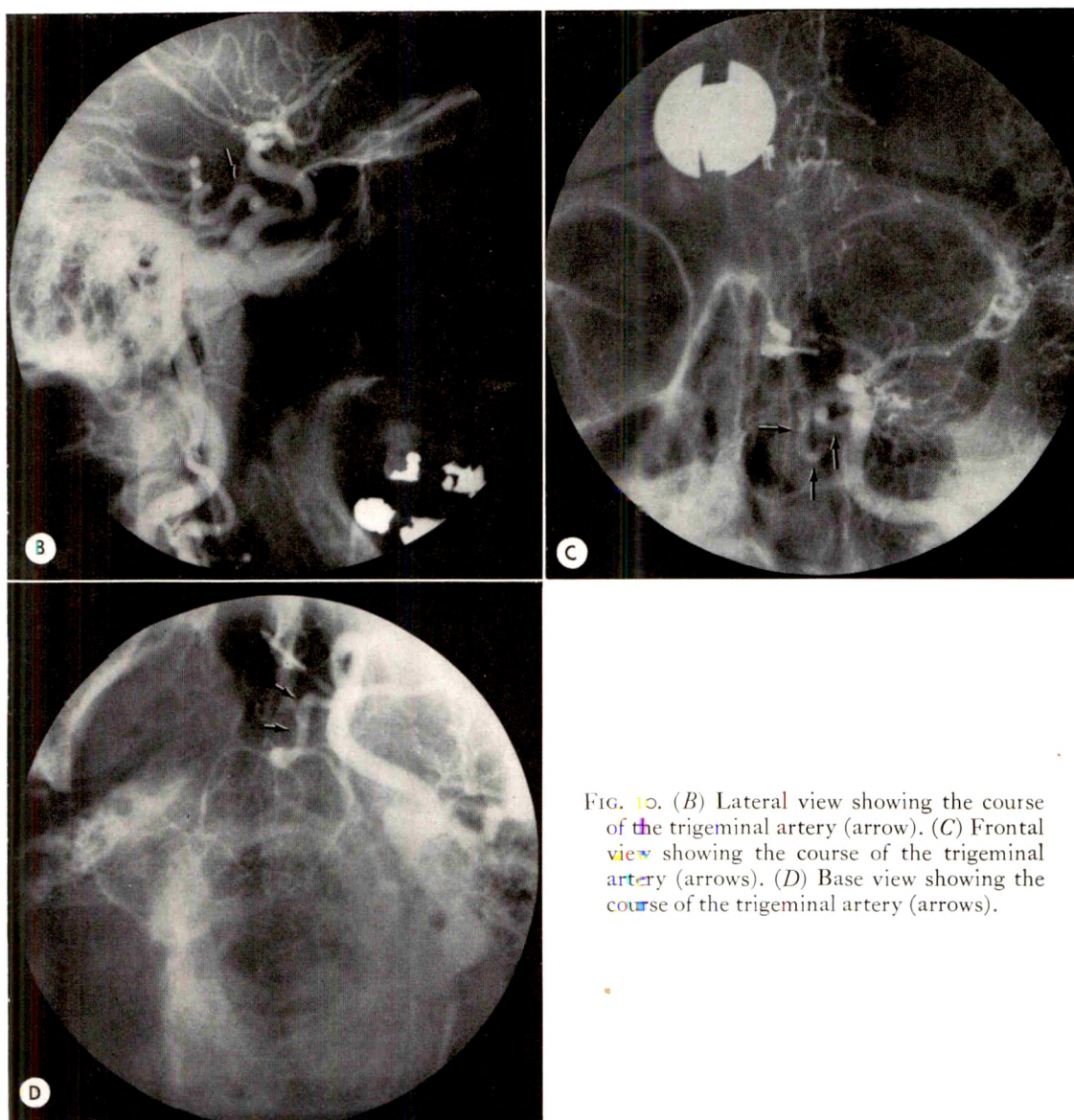


FIG. 10. (B) Lateral view showing the course of the trigeminal artery (arrow). (C) Frontal view showing the course of the trigeminal artery (arrows). (D) Base view showing the course of the trigeminal artery (arrows).



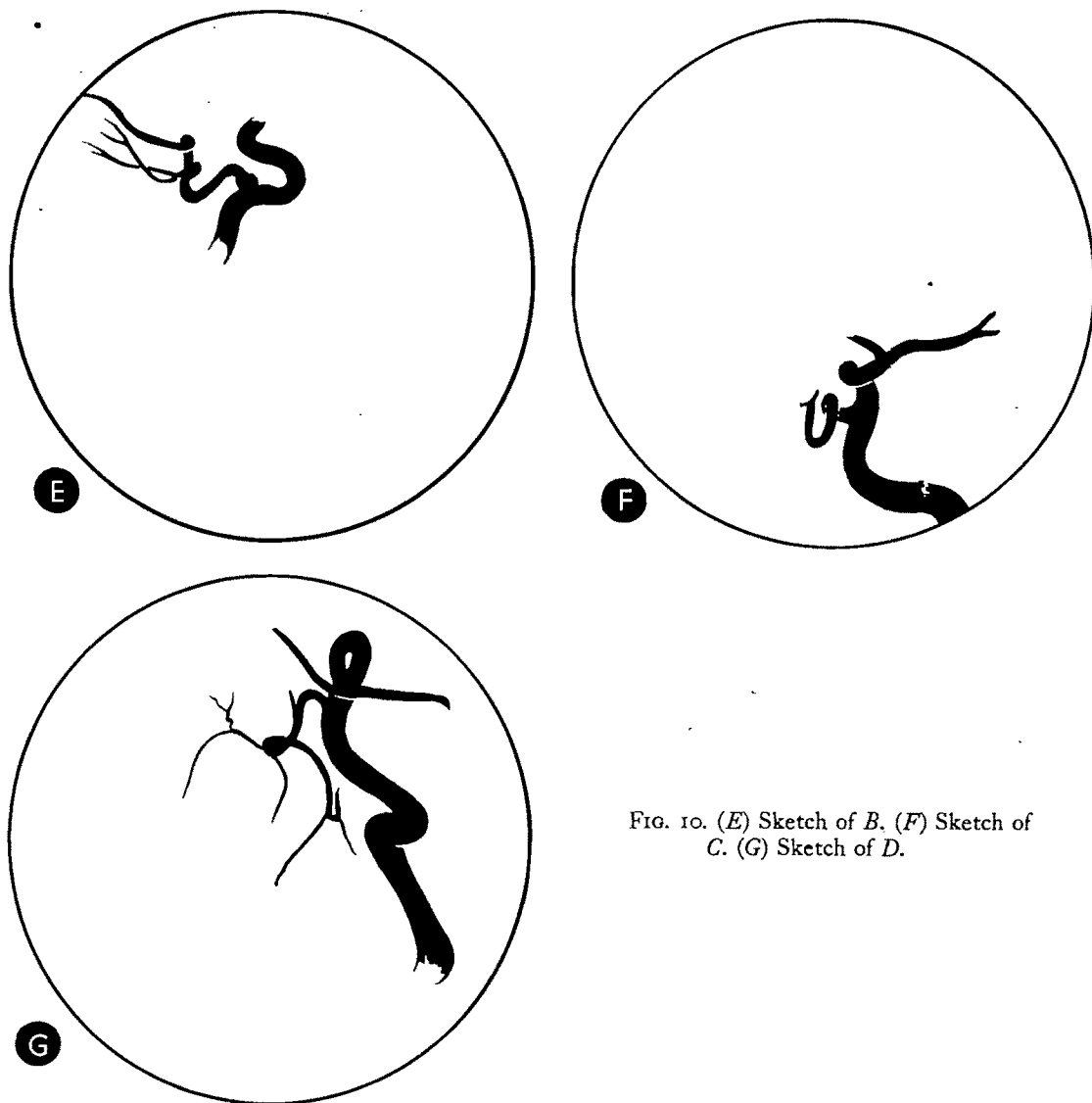


FIG. 10. (E) Sketch of B. (F) Sketch of C. (G) Sketch of D.

gliomas (Table 1). However, tentorial arteries have also been shown to supply gliomas and arteriovenous malformations. Interestingly enough, in this case the tumor, despite the demonstration of an enlarged tentorial artery, was relatively avascular at surgery.

CASE V. Left sided extradural mass. N.S., female, aged 47 years, complained of double vision following a blow to the right side of the head 9 months before admission. This resolved spontaneously. Six weeks before admission, double vision recurred. On examination, she

had a partial right third nerve palsy. A right carotid arteriogram (Fig. 7, A and B) showed forward and downward displacement of the vertical and horizontal segments of the cavernous internal carotid artery. The meningo-hypophyseal trunk passed medially and upwards. The hypophyseal and tentorial branches were stretched and displaced upwards; a short segment of the dorsal meningeal branch was noted. The proximal portion of the basal vein also showed upward displacement. Vertebral angiography showed posteromedial displacement of the proximal posterior cerebral artery. Surgery revealed a large cystic mass in the floor of

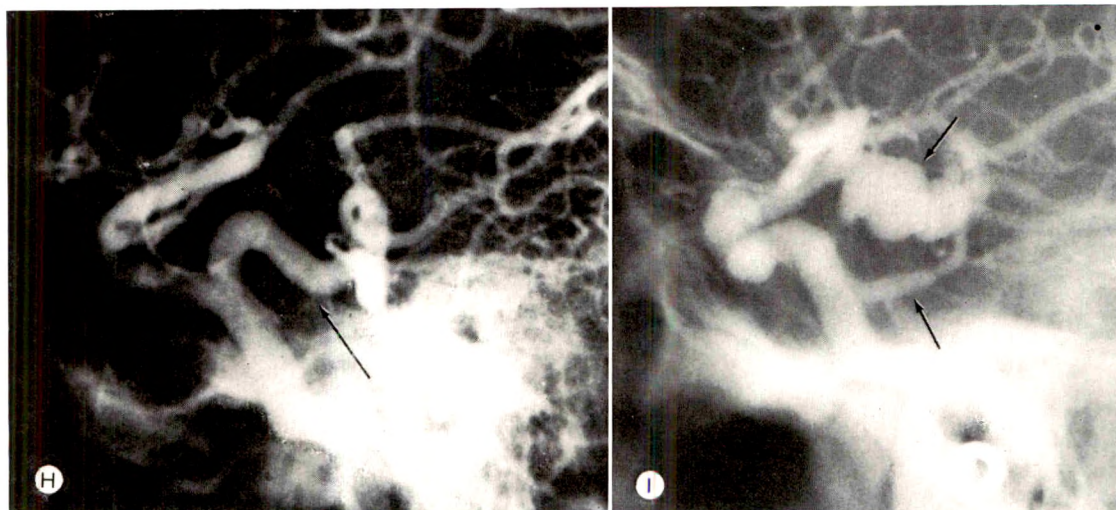


FIG. 10. (H) Large trigeminal artery (arrow). (I) Trigeminal artery (lower arrow) and internal carotid artery aneurysm (upper arrow).

the middle fossa, which was extradural. On aspiration with a No. 22 needle, blood was obtained. No biopsy was taken.

*Comment.* The angiographic appearances were those of an extradural mass, possibly a fifth nerve neuroma or a chordoma. This is an example of "unveiling" of the meningo-hypophyseal trunk. The main trunk passed upwards and medially while the hypophyseal and tentorial branches were displaced upwards.



FIG. 11. Pituitary tumor. Carotid arteriogram showing meningo-hypophyseal trunk (arrow).

CASE VI. Hypothalamic glioma. R.P., male, aged 6 years. Admitted with history of sexual precocity. Bone age was estimated at 10 to 11 years. Ketosteroid levels were in the lower adult range. An air encephalogram (Fig. 8A) outlined a mass in the region of the hypothalamus. Carotid arteriograms (Fig. 8, B-E) demonstrated a prominent inferior hypophyseal artery which showed lateral and downward displacement in the interpeduncular cistern.

*Comment.* This is the first occasion that we have seen an inferior hypophyseal artery supplying a tumor of the hypothalamus, although we have searched for this vessel. In this instance, the size of the mass and the displacement of the vessels account for its being visualized.

#### SUMMARY

A discussion of the roentgenology of the meningo-hypophyseal trunk is presented, along with illustrative case examples.

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The authors express their appreciation to Drs. J. D. Miller, L. C. Grisdale and T. J. Speakman for permission to include Case VI; to Mr. M. Meyers for the line drawings; to the



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## TENTORIAL BRANCH OF THE INTERNAL CAROTID ARTERY (ARTERIA TENTORII)\*

### REPORT OF THREE CASES

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IN 1956 Bernasconi and Cassinari<sup>1</sup> demonstrated by roentgenography a small artery supplying tentorial meningiomas in 5 out of 7 cases verified at operation. They suggested that this artery arose from the external carotid and considered it to be a pathognomonic sign of tentorial meningioma.

Although the artery was thereafter named "the artery of Bernasconi and Cassinari," similar angiographic evidence had been reported by Huber<sup>6</sup> a few years prior to their article.

In a paper read at the Fifth Symposium Neuroradiologicum in Bruxelles, Wickbom and Stattin<sup>11</sup> also described a vessel which took off from the carotid siphon, passed backwards to the tentorium and supplied a tentorial meningioma with blood. In the same year Krayenbühl and Yasargil<sup>7</sup> described an artery supplying a subtentorial arteriovenous malformation. Although the authors described this artery as the primitive trigeminal artery, their illustration clearly showed it to be the tentorial branch of the internal carotid artery.

In 1960, Frugoni *et al.*<sup>4</sup> described a similar artery in 8 cases of tentorial meningiomas, 2 cases of meningiomas of the falx, and 1 case of parasagittal meningioma. In the cases of Bernasconi and Cassinari and of Frugoni *et al.*, the contrast medium was injected into the common carotid artery, and the exact origin of the artery was not visualized. In 1 case of Frugoni *et al.*, however, the vessel did not fill when the external carotid artery was selectively punctured, and the authors cast some doubts on Bernasconi and Cassinari's view of the external carotid origin of this vessel.

In 1961, Stattin<sup>9</sup> described a vessel which

arose from the intracavernous portion of the carotid siphon in 8 out of 10 cases with tentorial meningiomas examined by selective internal carotid arteriography. They observed similar vessels in 7 more miscellaneous cases: 1 with arteriovenous malformation in the pineal region, 1 with the vascular malformation near the anterior attachment of the tentorium, and the remaining 5 cases with cerebral thrombosis or aneurysm.

Frugoni *et al.*,<sup>5</sup> in their further article, finally proved the internal carotid origin of the so-called Bernasconi Cassinari artery, and added several cases with arteriovenous malformation in the occipital region. Cortes *et al.*<sup>3</sup> described 4 instances of tentorial arteries, 1 in a patient with malignant glioma with invasion of the tentorium and 3 in patients with arteriovenous malformation in the tentorial region. Taveras and Wood<sup>10</sup> also described a case with tentorial meningioma, and Krayenbühl and Yasargil<sup>8</sup> illustrated their excellent monograph with 5 cases of the arteriae tentorii, 2 with tentorial meningiomas, 1 with angioma in the tentorial region, 1 with temporo-occipital basal glioblastoma and the remaining 1 with the metastatic carcinoma in the temporo-occipital region. Also, Bernasconi, Cassinari and Gori<sup>2</sup> recently reported a tentorial artery demonstrated in a case of falco-tentorial angioma.

#### REPORT OF CASES

CASE I. Y.N., a 38 year old female, was admitted to our clinic in 1962, with a 4 month history of paresthesia of the left half of the face and body, and diminished vision and tinnitus on the left side.

Left carotid arteriography was performed. The infra- and supraclinoid portions of the in-

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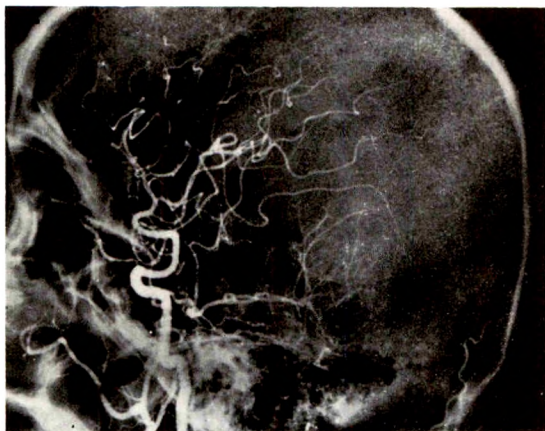


FIG. 1. Case I. Preoperative left lateral carotid angiogram. Note the arteria tentorii supplying the tumor stain (tentorial meningioma).

tential carotid artery were normal, and the anterior cerebral artery was not filled from the left side.

The frontal roentgenogram showed a slight elevation of the horizontal part of the middle cerebral artery, medial displacement of its knee, and a sharply demarcated and round abnormal vascular stain superimposed on the left orbita. An artery, 1 mm. in diameter, branched off from the cavernous portion of the internal carotid and supplied the stain.

In the lateral roentgenogram the anterior choroidal artery was elevated and the posterior communicating artery was not filled. The middle cerebral artery and its branches were elevated. A round tumor stain was demonstrated above the mastoid cells, and the tortuous vessel, which arose from the cavernous portion of the internal carotid, passed posteriorly and supplied the tumor stain together with the meningeal vessels of the external carotid origin (Fig. 1). The tumor stain was sharply demarcated, showing a sun-burst appearance, approximately 5 cm. in diameter, but early draining of the tumor was not observed.

A total removal of the tentorial meningioma (72 gm.) was performed, and the patient was discharged.

Six months later, the patient was readmitted complaining of a fever. A repeat arteriography was nonremarkable. The tortuous branches of the internal carotid artery, which had supplied the tumor stain, were again demonstrated, but the tumor stain was absent (Fig. 2). The previous operative site was re-explored and a brain

abscess was drained. The patient responded well and was discharged.

A few months later, the patient died suddenly in status epilepticus. Permission for autopsy was not granted.

CASE II. I.S., a 58 year old male, was admitted to our clinic with complaints of headache, blurred vision, and tinnitus and loss of hearing on the right side. Further positive neurologic findings on admission were: bilateral choked disks; right trigeminal, facial and glossopharyngeal palsies; and cerebellar ataxia.

Pneumoencephalography revealed displacement of the third ventricle to the left, and a marked elevation of the floor of the third ventricle.

Right carotid arteriography was performed. In the lateral view, the middle cerebral artery and its branches were elevated, and an abnormal artery, which arose from the posterior part of the cavernous portion of the internal carotid artery, passed posteriorly and supplied a tumor stain. The draining vein was not filled (Fig. 3). The surgical specimen was diagnosed as meningioma.

CASE III. S.O., a 31 year old male, was admitted twice, in 1957 and 1963. Each time a subtotal resection of the glioblastoma in the left temporal lobe was performed, and the patient was discharged after radiation therapy.

One month prior to the third admission in 1964, the patient noticed failing vision, nausea and swelling of the operative site in the left temporal region.

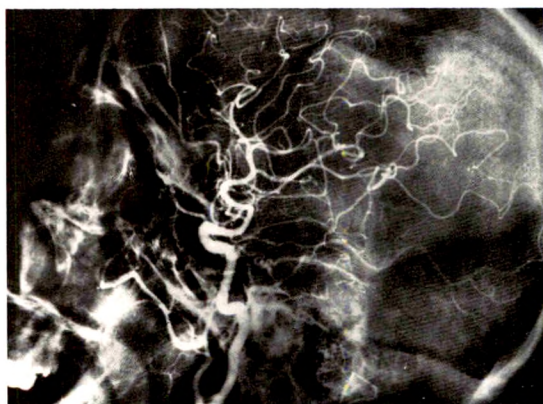


FIG. 2. Case I. Postoperative left lateral carotid angiogram. The arteria tentorii is still filled, but the tumor stain is not visualized.



Left carotid arteriography was performed and contrast medium was injected into the internal carotid artery.

The frontal projection disclosed medial displacement and stretching of the supraclinoid portion of the internal carotid artery with elevation of the bifurcation. The horizontal portion of the middle cerebral artery was markedly elevated. The anterior cerebral artery showed no shift across the midline and the anterior choroidal artery was displaced medially, describing a wide semicircle.

The lateral roentgenogram showed a marked elevation of the middle cerebral artery and its branches. The sylvian triangle was deformed and elevated, particularly in its posterior part. The supraclinoid portion of the internal carotid artery was elevated and displaced forward. The anterior choroidal artery was enlarged.

A tumor stain, approximately 5 cm. in diameter, was noted in the anterior two-thirds of the temporal lobe. Both frontal and lateral projections showed an unusual artery with a beaded appearance which was seen to supply the tumor. The artery arose from the posterior end of the cavernous portion of the internal carotid artery, but the anterior choroidal artery and small branches of the middle cerebral artery also contributed to the blood supply of the tumor (Fig. 4).

The patient improved and was discharged after subtotal removal of the recurrent temporo-occipital basal glioblastoma and the placement of a ventriculo-atrial shunt.

#### COMMENT

Reviewing the available literature, it seems certain that the vessel described by

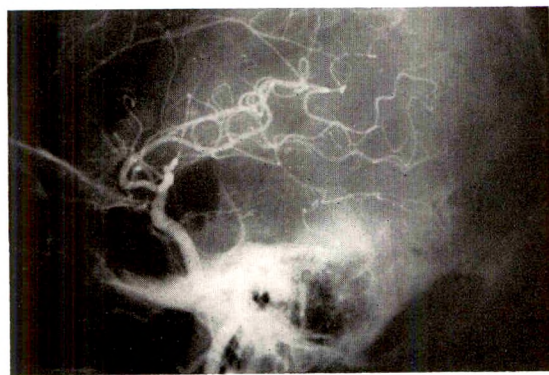


FIG. 3. Case II. Right lateral carotid angiogram showing the tentorial meningioma.



FIG. 4. Case III. Preoperative left lateral carotid angiogram. Note the arteria tentorii supplying the tumor (temporo-occipital basal glioblastoma multiforme).

Bernasconi and Cassinari arises from the carotid siphon just distal to the entrance of the internal carotid artery into the cavernous sinus, and not from the external carotid artery. The artery passes backwards, reaches the tentorial attachment and further courses alongside the free edge of the tentorium.

In 2 Macaque monkeys, the cervical carotid artery was injected with the colored neoprene latex under pressure, and the head was fixed in toto in 10 per cent formalin solution for 1 week.

A meticulous dissection of the specimen disclosed the small artery which arose from the posterior part of the cavernous portion of the internal carotid artery, and passed backwards. The artery sent several tiny branches to the gasserian ganglion and its meningeal coverings, and reached the tentorial attachment. The artery supplied the free edge of the tentorium, falco-tentorial attachment and their neighboring structures, and anastomosed with the corresponding vessel on the other side and with the meningeal vessels of the external carotid and vertebral origin (Fig. 5).

In man, the internal carotid artery gives rise to several small branches in its intracavernous portion.<sup>9</sup> Among those, the course of the *arteria tentorii* is in good accordance with the roentgenologic findings





FIG. 5. Tentorial branch of the internal carotid artery in the Macaque monkey. Dissection after intra-carotid injection of colored neoprene latex.

of the Bernasconi Cassinari artery and also with the anatomic findings in the monkey.

Although such tiny branches of the internal carotid artery cannot be visualized roentgenographically under usual conditions, it is quite reasonable to assume that the vessel becomes enlarged and more easily visualized in cases of tentorial meningioma.

However, visualization of the Bernasconi Cassinari artery is by no means pathognomonic of meningioma of the tentorium. It is seen also in meningiomas of the posterior falx, falco-tentorial arteriovenous malformations, temporo-occipital gliomas and other situations. The artery indicates the location and rich vascular supply of the lesion and not the type of pathology of the lesion.

#### SUMMARY

1. The arteria tentorii (Bernasconi and Cassinari's artery) was visualized by carotid angiography in 3 cases of brain tumors, 2 with tentorial meningiomas and the other with a temporo-occipital basal glioblastoma multiforme.

2. The available literature is reviewed and the anatomic studies in the monkey are described briefly.

3. In the majority of the cases reported, the arteria tentorii was associated with tentorial meningioma; but visualization of

the artery is by no means pathognomonic of tentorial meningiomas. It only indicates the site and the rich vascularization of the lesion.

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## ASCENDING PHARYNGEAL-VERTEBRAL ANASTOMOSIS\*

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THE demonstration of collateral pathways in occlusive disease of the vertebral and carotid arteries has become commonplace in patients with cerebrovascular insufficiency.

Schechter<sup>4</sup> has emphasized the importance of occipital-vertebral anastomosis. Bosniak<sup>1</sup> has described a variety of collateral pathways, called collectively the "cervical arterial collateral network." These involve the occipital branch of the external carotid artery, the muscular branches of the vertebral artery, and the thyrocervical and costocervical trunks of the subclavian artery. Collateral circulation has been shown to occur across the midline via the muscular branches of the vertebral arteries by North *et al.*,<sup>3</sup> and through the superior thyroid and lingual arteries by Tatelman.<sup>5</sup>

Wiedenmann<sup>6</sup> in 1962 described an anastomosis of the ascending pharyngeal artery, a branch of the external carotid artery, with the vertebral artery in a patient with no evidence of occlusive disease. This pathway was only shown because of accidental puncture of the ascending pharyngeal artery followed by injection of contrast medium. He felt that opacification of the pathway was due to increased pressure gradient by injection in this small vessel. To our knowledge, this collateral channel has yet to be described in a patient with definite occlusive vascular disease. The demonstration of this collateral in such a patient prompts this report.

### REPORT OF A CASE

A 56 year old male with symptoms of intermittent but progressive cerebrovascular insufficiency manifested by four "small" cerebrovascular accidents involving the right side of

the body was examined by left carotid arteriography. This was performed by direct puncture of the left common carotid artery. The internal carotid artery was occluded at its origin. The vertebral-basilar artery and its branches were opacified via collateral circulation between the occipital artery and the vertebral artery (muscular branches), but also via the ascending pharyngeal artery, and the vertebral artery at the level of C 2 vertebral body (Fig. 1, A and B). The anterior cerebral circulation was filled via the posterior communicating artery. This would indicate that the blood supply of the left cerebral hemisphere and the structures of the posterior fossa are dependent mainly on the anastomosis of the ascending pharyngeal artery and the vertebral artery.

The ascending pharyngeal artery is the smallest branch of the external carotid artery arising on the posteromedial aspect of the external carotid artery just above its origin. It then courses cephalad between the internal carotid artery and the pharynx to the base of the skull. Descending branches of this vessel supply the pharynx. Other branches are the prevertebral branches which anastomose with the vertebral artery, the inferior tympanic artery, and meningeal branches<sup>2</sup> (Fig. 2 and 3).

**Comment.** This case proves Wiedenmann's theory that this vessel, in patients with carotid or vertebral artery disease, is a potential source of blood supply to the brain. The case is of further interest in that it shows two other potential sources of blood supply to the brain; namely, the occipital-vertebral channel and reverse flow through the posterior communicating artery to the anterior cerebral circulation.

### SUMMARY

A case is presented of a patient with internal carotid artery occlusion showing a

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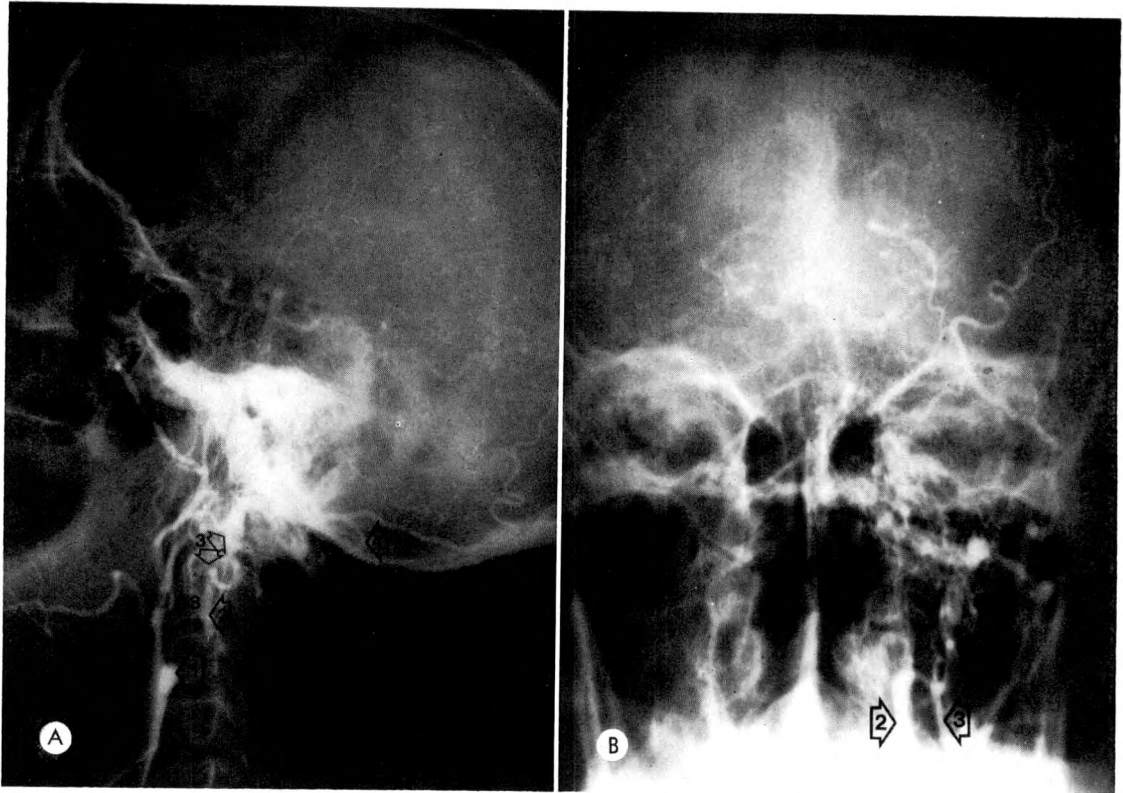


FIG. 1. (A) Lateral carotid angiogram showing occlusion of the internal carotid artery (1). The vertebral artery (2) is opacified by the ascending pharyngeal artery and its branches (3). The occipital-vertebral anastomosis is seen (4). (B) Anteroposterior view showing the vertebral artery (2) and the ascending pharyngeal artery (3).

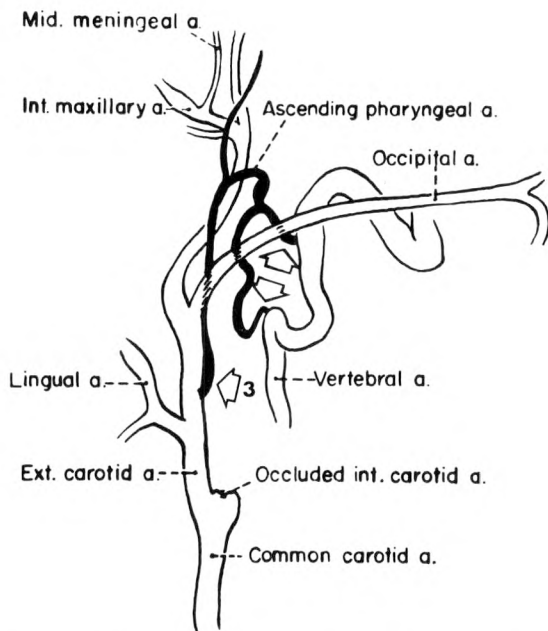


FIG. 2. Schematic drawing of Figure 1A showing the ascending pharyngeal-vertebral anastomosis in black.

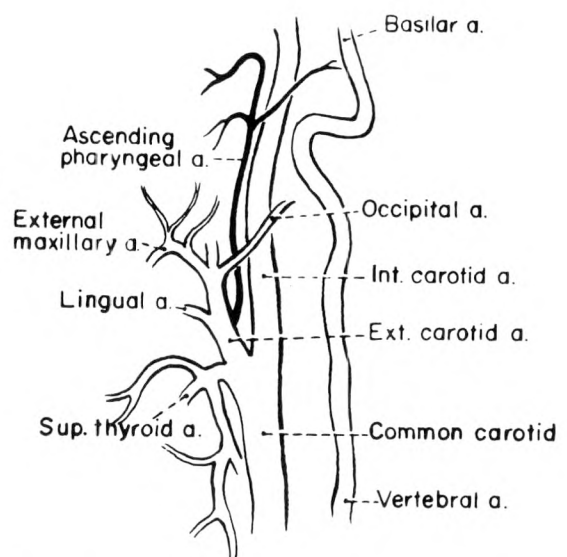


FIG. 3. Modified from Gray's Anatomy, 26th Edition, page 636, showing the normal relationships of the ascending pharyngeal artery and carotid vasculature.

heretofore theoretic collateral channel from the external carotid artery to the vertebral artery via the ascending pharyngeal artery.

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## THE CLINICAL SIGNIFICANCE OF THE AZYGOS ANTERIOR CEREBRAL ARTERY (A.C.A.)\*

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WITH current augmented usage of cerebral angiography, it is important to emphasize the anomalies of the cerebral circulation, as they are not rare and may have profound clinical implications. Variations of the anterior cerebral artery (A.C.A.), in particular, deserve attention because they govern the distribution of blood to the two hemispheres and certain clinical syndromes of cerebral vascular occlusive disease will be a function of their specific anatomic arrangement. Occasionally, both cerebral hemispheres are supplied by a single A.C.A. Since this anatomic variation is not always readily discernible roentgenographically when only 1 carotid artery is examined, careful observations were made of all carotid arteriograms done during the past year for examples of unpaired A.C.A.s to correlate their demonstration with the presence or absence of significant clinical abnormalities.

### PHYLOGENY

A proper appreciation of the A.C.A. aberrations requires cognizance of the phylogenetic development of the vessel. Critchley<sup>8</sup> and Watts,<sup>12</sup> over 30 years ago, summarized the pertinent contributions of preceding investigators (Rothmann,<sup>9</sup> Windel,<sup>18</sup> and Lesem<sup>7</sup>) and re-emphasized the close association of the type of A.C.A. with the pattern of the anterior portion of the circle of Willis.

The most primitive pattern occurs in fishes, reptiles, amphibians, and birds. In these animals no anterior communicating artery exists, so there is no real circle of Willis and the two A.C.A.s lie parallel, without communication, on the mesial aspect of the olfactory lobe. In snakes, tortoises, and crocodiles, however, the

A.C.A.s may unite to form a common midline vessel which has been called the "azygos (unpaired) artery." The common mammalian pattern (rabbit, squirrel, pig) consists of no anterior communicating artery; instead 2 short A.C.A.s are present which converge and form a median azygos artery which may or may not bifurcate more distally.

Among the lower primates, there is no anterior communicating artery and the anterior portion of the circle of Willis is quite variable. The A.C.A.s of the chimpanzee unite at the entrance of the longitudinal cerebral fissure and course forward to the corpus callosum rostrum where the trunk then divides. The situation is quite similar in monkeys. The orangutan usually has 2 A.C.A.s, although occasionally 3 have been found. The arterial anatomy of the gorilla most closely resembles that of man.

Lesem<sup>7</sup> stated that in the human fetal and infantile brains which he examined he found the sequences marking the transition of the A.C.A. from a single to 2 arteries. In 22 out of 32 human fetuses (68 per cent) and in 18 out of 21 infant brains (75 per cent), the 2 A.C.A.s united to form a single artery within the longitudinal fissure. DeVriese<sup>4</sup> also found a higher incidence of azygos anterior cerebral arteries in the fetus than in the adult and advanced the hypothesis that originally, during an early stage of embryonic life, either 1 or 3 anterior cerebral arteries could exist. If only 1 existed, the single stem would duplicate and, in the case of 3 vessels, 1 would degenerate.

Baptista<sup>1</sup> recently reviewed the literature and found that of 2,153 brains studied, 23 had unpaired or azygos A.C.A.s. In his own series of 381 brains, there was only 1 un-

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paired A.C.A. He distinguishes the entity of azygos A.C.A. from bihemispheric A.C.A., the latter representing a situation in which 1 A.C.A. is dominant and bifurcates at a variable distance from its origin to supply both hemispheres. This latter condition he found in 45 of his series. Many of the arteries that other authors have called "azygos A.C.A.s" because they were seen as single, large main vessels ascending toward the corpus callosum, have been called "bihemispheric A.C.A.s" by Baptista if they derived their main blood supply from 1 carotid artery.

#### THE COMMON ANATOMY OF THE A.C.A. IN HUMAN

The anterior cerebral artery arises from the internal carotid artery distal to the carotid syphon, crosses the anterior perforating substance, where it gives off small medial striate vessels, and reaches the longitudinal intercerebral fissure. At this point, it connects with the contralateral anterior cerebral artery by the small anterior communicating artery. It then turns upwards in the direction of the genu of the corpus callosum with which it is intimately associated as it parallels it in a wide arc to

continue over the corpus callosum to terminate near the splenium. It may lie in the sulcus of the corpus callosum or on the medial surface of the hemisphere; occasionally, it lies in the cingulate sulcus. The left and right A.C.A. are parallel, with the left usually residing slightly posterior.

The first set of side branches, the *medial striate* arteries, consists of 3 or 4 very thin perforating branches, 1 of which may distinguish itself by a wider caliber and has been called the recurrent artery of Heubner. This vessel runs in a lateral and dorsal direction and, finally, penetrates into the lateral part of the anterior perforating substance.

The major branches of the distal A.C.A. are shown in Figure 1. The branches are variable and 2 or 3 of them may issue from a common stem. The first is the *orbital* artery (prefrontal artery of Critchley<sup>3</sup>). This begins just beyond the anterior communicating artery. It may arise as a separate stem, as a common stem with the frontopolar or, rarely, as a common stem with the frontopolar and callosomarginal arteries. It courses forward and downward to reach the orbital side of the frontal lobe under the orbital and olfactory gyri.

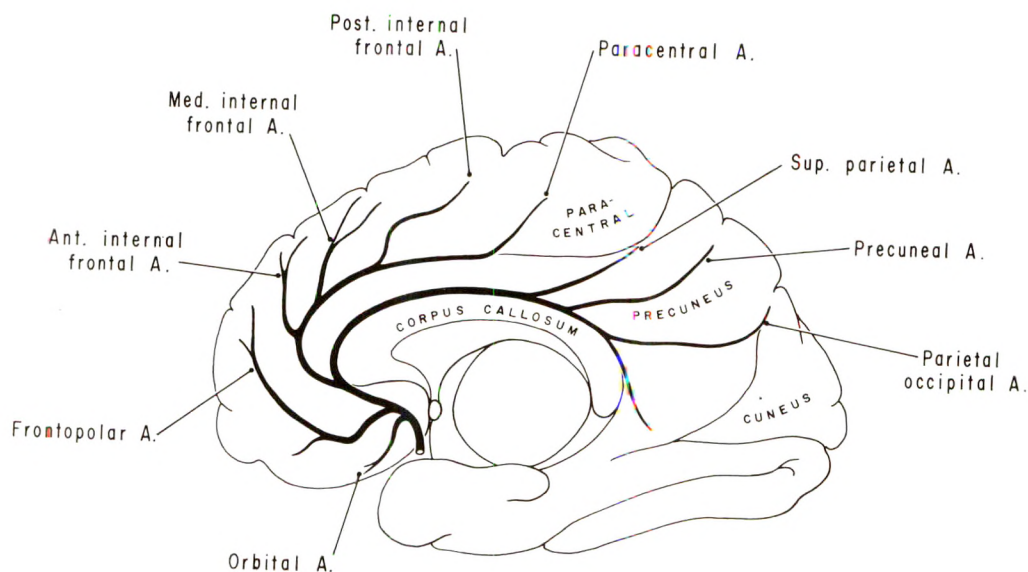


FIG. 1. Scheme of the main branches of the anterior cerebral artery (A.C.A.).



The second in the series of the distal A.C.A. branches is the *frontopolar* artery. This begins on the convex side of the A.C.A. below the genu of the corpus callosum. It may have a separate origin, or arise from a common stem with either the orbital artery or the anterior internal frontal artery. It curves slightly downwards, enters the supraorbital sulcus and divides over the anterior portion of the superior frontal gyrus which it supplies.

The next branch of the A.C.A., the *callosomarginal* artery, is by far its largest branch and usually arises in the region of the genu of the corpus callosum. It crosses the cingulate gyrus, passes through the cingulate sulcus and during its course gives off successively the anterior, medial, and posterior internal frontal arteries and often the paracentral artery. The *anterior internal frontal* artery begins near the genu, has a short ascending course, and supplies the antero-medial portion of the superior frontal gyrus on the convex and interhemispheric aspects. The *medial internal frontal* artery originates next and divides over the posteromedial portion of the superior frontal gyrus. The *posterior internal frontal* artery supplies the posterior part of the superior frontal gyrus on the convex and interhemispheric aspects. The *paracentral* artery ascends to reach the paracentral lobule and supplies it and the adjoining superior parts of the precentral and postcentral gyrus.

After the anterior cerebral artery has given off the callosomarginal artery, it continues as the *pericallosal* artery, which often provides 1 or 2 small branches to the posterior central cortex, and *superior parietal* convolution and then as the more prominent *precuneal* artery, which courses in a posterior arc over the cingulate gyrus to reach the precuneus. It perfuses most of the precuneus and the superoposterior part of the superior parietal convolution.

The pericallosal artery may terminate in the precuneal branch or may continue to a *parietal occipital* branch which runs backward and ends in the parieto-occipital fissure. The terminal portion of the pericallosal artery is often referred to as the

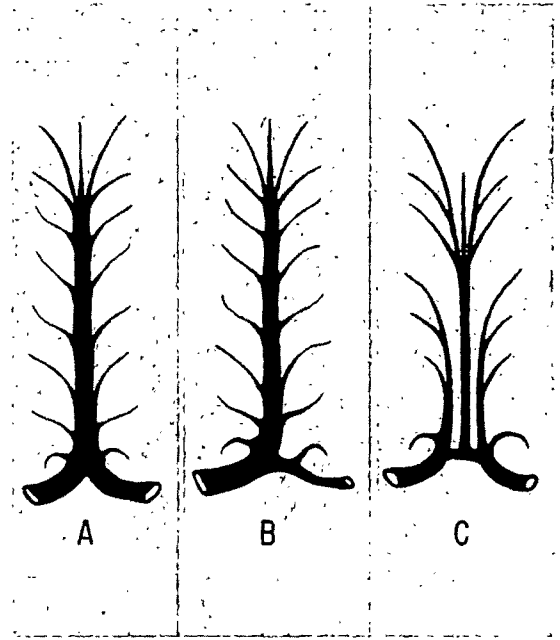


FIG. 2. Anomalies occurring in the distal portion of the A.C.A. (A) True azygos A.C.A. All the major branches arise from a single trunk. (B) Bi-hemispheric A.C.A. Most of the major vessels arise from a single A.C.A. (C) Triple A.C.A. An accessory vessel arises from the anterior communicating artery which may be bi-hemispheric.

*posterior pericallosal* artery. This gives off small branches to the cingulate gyrus and penetrating vessels to the corpus callosum and then continues in the sulcus of the callosum to supply the upper two-thirds of the splenium and anastomose with the posterior cerebral artery.

Considerable variation occurs in the pattern of the A.C.A. and these have been described in detail by Baptista.<sup>1</sup> He described 3 types of anomalies occurring in the distal A.C.A. (Fig. 2): (1) a single large vessel giving off all major vessels to both hemispheres; (2) 2 A.C.A.s, but only 1 supplying the majority of vessels to both hemispheres (a bi-hemispheric vessel); and (3) 2 A.C.A.s plus an additional median vessel (an accessory A.C.A. may be bi-hemispheric or go only to one hemisphere).

#### ROENTGENOGRAPHIC APPEARANCE OF THE A.C.A.

Brief mention of the variations of the

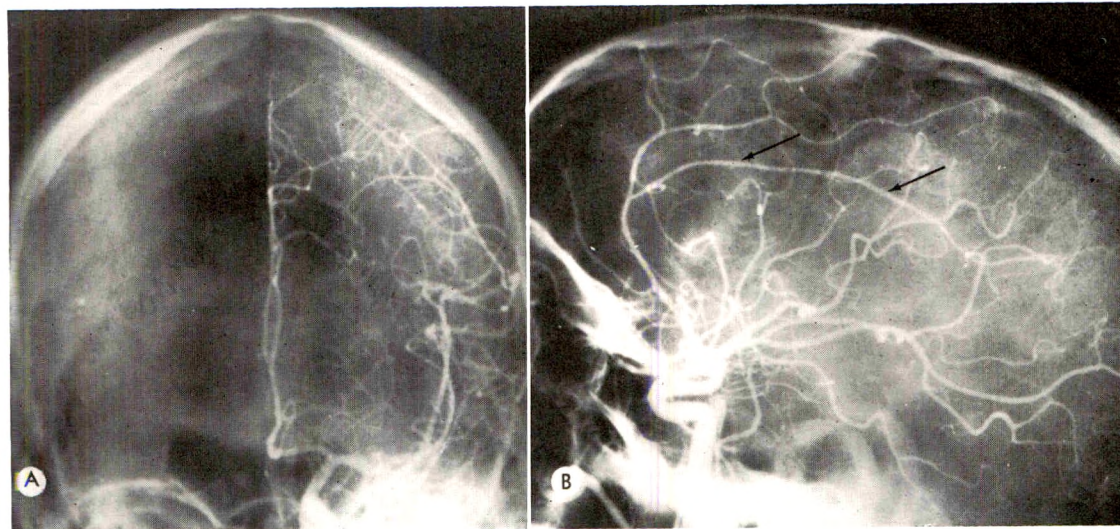


FIG. 3. Left carotid arteriograms. (A) Anteroposterior view. The A.C.A. vessels supply only one hemisphere. (B) Lateral view. No pericallosal artery is filled. Arrows point to the callosomarginal artery.

A.C.A. has been made by Taveras and Wood.<sup>11</sup> The most common irregularity is a difference in size of the proximal portion of the 2 vessels, the one on the left more often being larger. At arteriography, both distal arteries frequently are filled from one side, especially when the left carotid artery is injected.<sup>10</sup> Occasionally, there is bilateral filling when both right and left carotid arteries are injected separately, probably

due to the union of the A.C.A.s for a short distance and the absence of a true anterior communication artery. This anomaly was noted in 3 of 30 brains dissected by Saltzman<sup>10</sup> and 6 of 200 brains studied by Windel.<sup>13</sup>

During fetal life, the anterior communicating artery gives rise to a branch which is called the median artery of the corpus callosum.<sup>2</sup> This usually atrophies but may

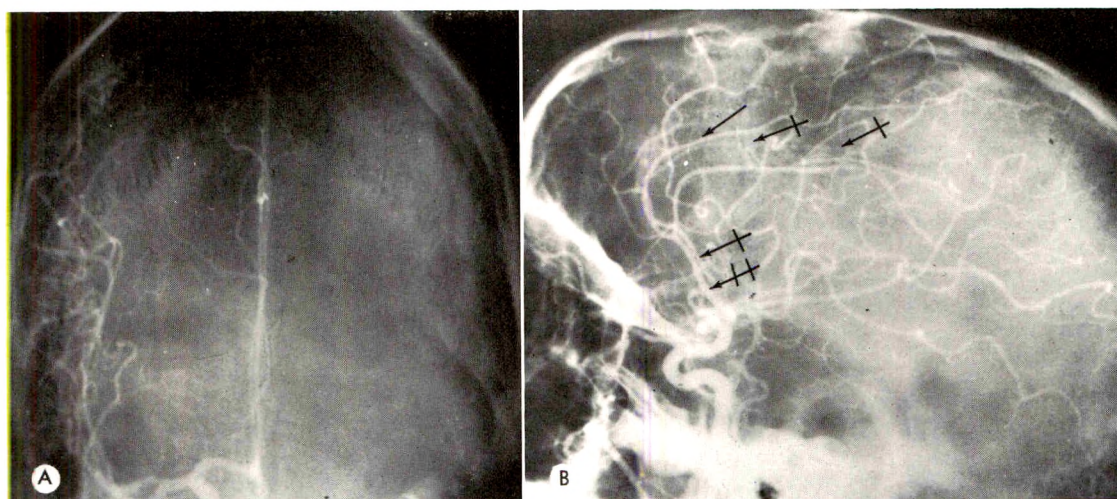


FIG. 4. Right carotid arteriograms. (A) Anteroposterior view. The A.C.A. sends vessels to both hemispheres. (B) Lateral view. Both pericallosal arteries are filled. The left pericallosal (↔) lies behind the right (↔) in its ascending portion, and then courses posteriorly in the region of the cingulate sulcus. The right callosomarginal artery is shown by the plain arrow (←).



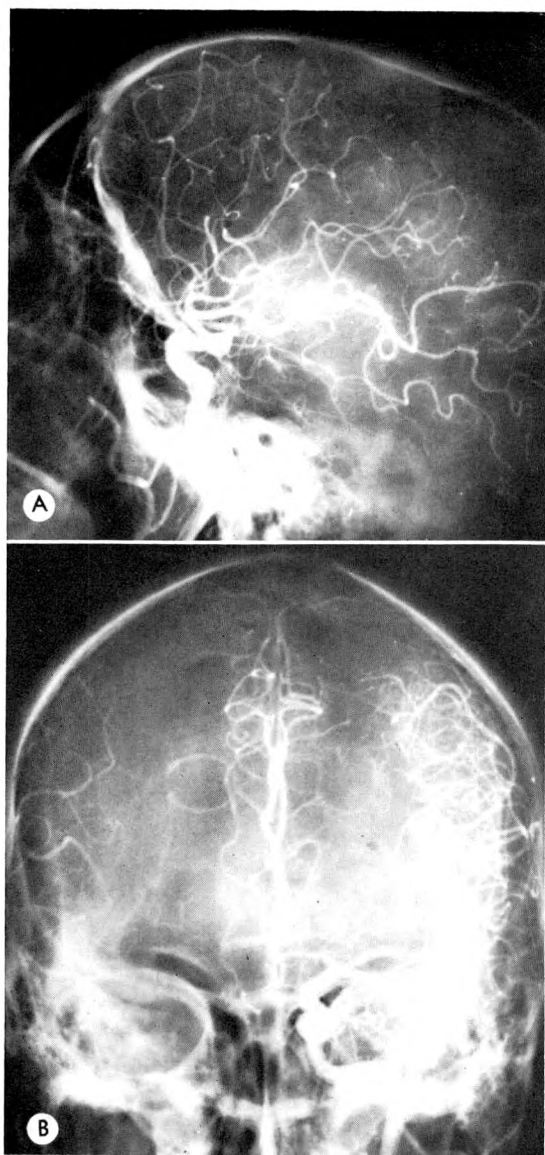


FIG. 5. (A) A left carotid arteriogram of a man suspected of having metastatic disease. No pericallosal artery is shown. The frontopolar and internal frontal arteries arise as a common branch. (B) An anteroposterior left carotid arteriogram taken with cross compression of the right carotid artery. A large right A.C.A. is filled, presenting as a large single midline vessel which supplies both hemispheres.

persist into adult life. Baptista found 1 in 135 brains he examined. They are difficult to differentiate roentgenologically from a frontopolar artery, or the opposite anterior cerebral artery if only one side is injected.

It is not always possible to be certain roentgenographically, even when there is simultaneous opacification of both internal carotid arteries, that a single large trunk represents a true azygos A.C.A. rather than an A.C.A. derived from one side and supplying both hemispheres with a rudimentary A.C.A. on the opposite side. Most authors reporting anomalies of the A.C.A. found at dissection, have commented upon finding unpaired, and triplicate A.C.A.s, but have not differentiated bihemispheric vessels. Figures 3, A and B; and 4, A and B show an example of a bihemispheric anterior cerebral artery. A left carotid arteriogram showed, in the anteroposterior view, an A.C.A. supplying one hemisphere. In the lateral view, its main continuing trunk was high and suggested the configuration of the pericallosal artery in hydrocephalus, but actually was a callosal marginal vessel in normal position. Injection of the right carotid artery showed the A.C.A. in the anteroposterior view to be bihemispheric and in the lateral view both pericallosal arteries originated from the right carotid artery. A more obvious bihemispheric vessel is shown in Figure 5, A and B.

To determine the angiographic frequency of A.C.A. anomalies, 107 consecutive carotid angiograms, in which bilateral opacification was obtained, were examined. In 95 cases, the normal A.C.A. distribution could be identified. There were 4 cases of azygos A.C.A. and 8 cases of triple A.C.A. or bihemispheric A.C.A. It is probable that upon anatomic dissection 1 or more of the cases classified as an azygos vessel would be shown by Baptista's classification to represent a bihemispheric artery. Figure 6, A and B shows 1 of the cases classified as having an azygos A.C.A. The patient was a man with a parietal lobe tumor and the azygos A.C.A. was an incidental finding.

It is not surprising that unpaired A.C.A.s occur with other congenital anomalies. Patients in whom the prosencephalon fails to divide into hemispheres, holotelencephaly, often have a single A.C.A.<sup>14,15</sup> One patient has been observed during the past year

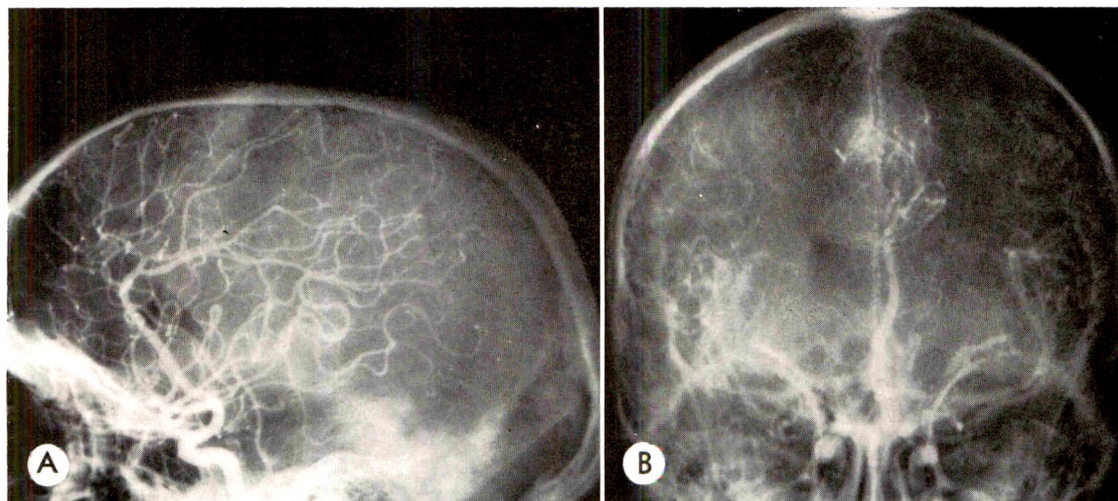


FIG. 6. (*A* and *B*) Right carotid angiograms with cross compression of a patient with a fronto-parietal tumor. The unpaired A.C.A. is an incidental finding.

(Fig. 7, *A* and *B*) with an arteriovenous malformation who had an azygos anterior cerebral artery which was verified at autopsy. Aneurysms of the pericallosal arteries, sometimes associated with malformations such as craniosynostosis and coarctation of the aorta, are not common. On the other hand, Laitinen and Snellman<sup>6</sup> reported 3 patients with a single A.C.A. trunk associated with aneurysms of the pericallosal arteries and Pool and Potts<sup>8</sup> state that the incidence of aneurysms of the pericallosal arteries is higher in patients with azygos trunks. Figure 8, *A* and *B*, is from a patient who entered the Peter Bent Brigham Hospital during the past year with a subarachnoid hemorrhage. Arteriograms demonstrated a single anterior cerebral artery and an aneurysm near its origin.

It is obvious that in cerebral vascular occlusive disease the location of an occlusion determines the extent and the severity of the ischemia. The following case history

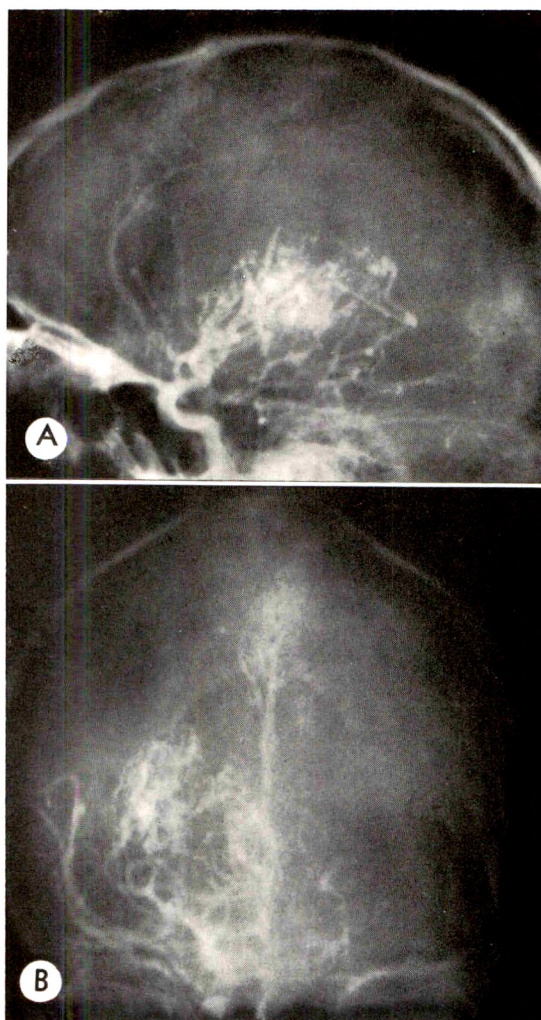


FIG. 7. (*A* and *B*) Right brachial arteriograms demonstrating a large arteriovenous malformation and what was shown at autopsy to be an azygos A.C.A. (Courtesy of Dr. Edward B. D. Neuhauser.)



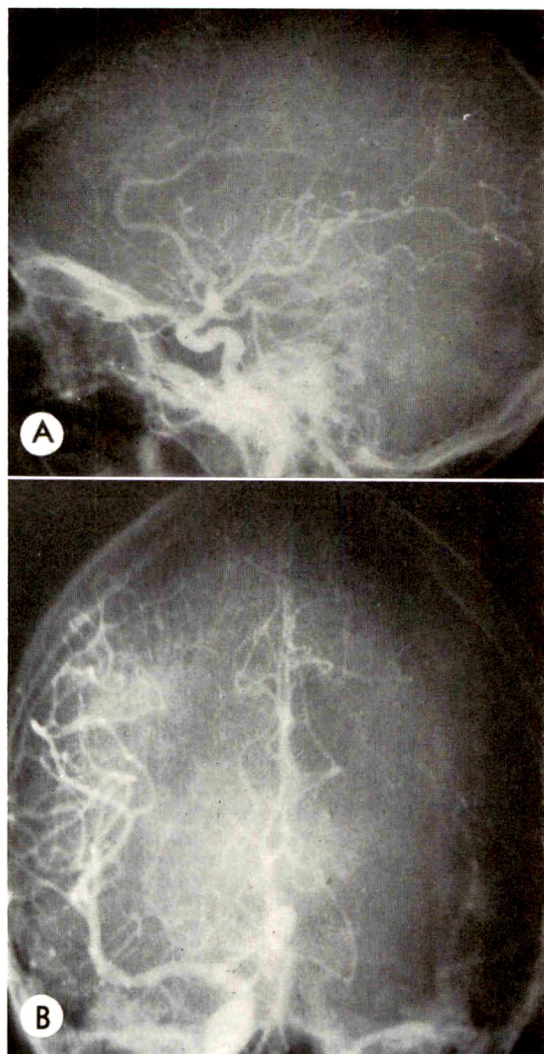


FIG. 8. (A and B) An azygos A.C.A. associated with an aneurysm near its proximal end.

dramatically illustrates the profound clinical implication of the azygos A.C.A. anomaly.

#### REPORT OF A CASE

A 48 year old woman file clerk (PBBH5-53-58) noted weakness of her left leg 1 month prior to admission, but was otherwise well until the day of admission when she experienced a grand mal seizure. She was incontinent of urine and feces and shortly thereafter became comatose. When admitted to a local hospital, her vital signs were normal, she was unresponsive to commands, she had no papilledema, but had a rigid left arm and leg. Deep tendon reflexes

were all exaggerated and she had ankle clonus. Soon after admission, she had another seizure, her blood pressure fell to 78/60 and she became cyanotic. She was treated with dilantin, decadron, and intravenous fluids and was transferred to the Peter Bent Brigham Hospital.

*Past history.* One year prior to admission, the patient had a hysterectomy, bilateral salpingo-oophorectomy and postoperative radiation therapy for mucinous cystadenocarcinoma of the ovary.

*Physical examination.* There were normal vital signs. The patient appeared dazed, but awake, and she was aphasic. The pupils reacted to light and there was no papilledema. There was a left central facial nerve palsy, a left hemiparesis with extreme spasticity and hyperreflexia, and positive Hoffmann and Babinski signs. She had no heart murmurs and physical examination was otherwise unremarkable.

*Roentgen examination.* Roentgenograms of the chest were normal as were the skull roentgenograms which showed a midline pineal gland and no fracture. Bilateral carotid arteriograms (Fig. 9, A, B and C) demonstrated no evidence of occlusion of any major vessel on the left. The left A.C.A. was small and extended only to the midline. On the right, several of the posterior insular branches of the middle cerebral artery were occluded. The A.C.A. was larger than the one on the left. It extended to the midline and fed a wide single midline vessel which was occluded just after giving rise to an orbital branch and another small branch which extended upward and laterally for a short distance. Part of the distal portion of a pericallosal vessel filled by anastomotic branches from the middle cerebral artery.

*Biopsy.* Supraclavicular lymph node biopsy revealed metastatic adenocarcinoma consistent with an ovarian primary lesion.

*Hospital course.* Shortly after admission, the patient developed Cheyne-Stokes breathing, hypotension, and appeared in extremis. She temporarily improved slightly on dexamethasone and intravenous fluids, but over the next 3 weeks she gradually deteriorated, developing cold, pulseless peripheral extremities in spite of normal blood pressures. A transient episode of fever disappeared on antibiotic therapy. There was a progressive thrombocytopenia with a low of 32,000. Terminally, the patient developed shallow respirations and finally, apnea. Post-mortem examination was unobtainable.



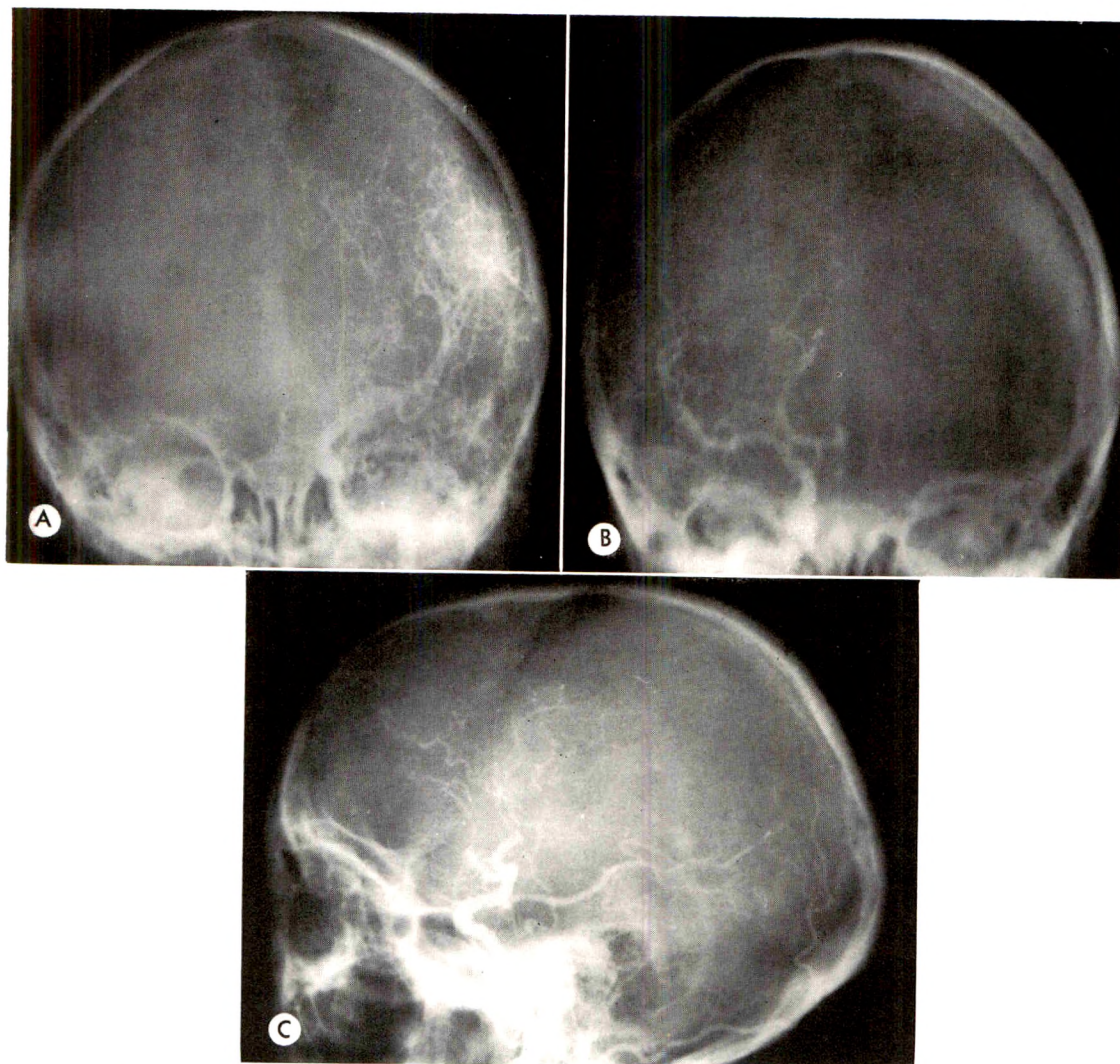


FIG. 9. (A) Left carotid arteriogram. A small A.C.A. extends only to the midline. No pericallosal artery fills. (B) Right carotid arteriogram. A single, midline A.C.A. fills and is occluded beyond the origin of the orbital branch. (C) Right carotid arteriogram. Occlusion of the A.C.A. and most of the insular vessels. A large posterior cerebral artery comes directly from the carotid artery. On the original film the distal portion of the pericallosal artery was filled by anastomotic branches from the midline cerebral artery.

*Comment.* The nature of this patient's vascular occlusive disease was a clinical enigma. It was felt to be most likely non-bacterial thrombotic endocarditis with multiple peripheral emboli in a patient with metastatic disease. Also considered were a thrombotic thrombocytopenic phenomenon and metastatic carcinomatous emboli.

This case is unique in that it demonstrated occlusion of a single midline A.C.A. which resulted, most likely, in ischemic in-

farcction in both cerebral hemispheres and the corpus callosum.

#### SUMMARY

The phylogenetic development of the unpaired (azygos) anterior cerebral artery and its transient existence during human embryologic development are reviewed.

Retrospective examination of arteriograms of 107 patients in whom both internal cerebral arteries were opacified revealed



4 examples of azygos A.C.A.

Azygos A.C.A.s may occur with other congenital anomalies. Cases showing their occurrence with an arteriovenous malformation and an aneurysm of a pericallosal artery are presented.

The clinical implications of the azygos A.C.A. or bihemispheric anomaly, are illustrated by a case history of a patient who had occlusion of a single large anterior cerebral vessel.

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## PRIMARY ARACHNOID CYSTS OF THE SELLA TURCICA\*

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**I**NTRACRANIAL arachnoid cysts are well known entities but are not common. Primary arachnoid cysts involving the sella turcica are even more unusual, and we were able to find only 2 cases in the literature. The following case reports are considered warranted because of the rarity of this condition and the happenstance of having seen 2 cases in a 4 month period.

### REPORT OF CASES

**CASE I.** The first patient was a 17 year old white male student admitted because of a compression fracture of the first lumbar vertebra. Five years previously, the patient had sustained an orbital fracture but was otherwise in good health. Current examination was negative except for facial abrasions and lumbar tenderness. Roentgenograms of the skull and facial bones showed an old well-healed fracture of the inferior rim of the left orbit and an enlarged, eroded sella turcica. Roentgenograms from the time of the original facial injury were obtained and the sella turcica was seen to have been eroded at that time with no change over the intervening 5 years.

Visual fields and pituitary function studies were normal as was the spinal fluid.

Bilateral carotid angiograms showed questionable elevation of the anterior cerebral artery on the right, and slight elevation of the venous angle, bilaterally, consistent with suprasellar extension of pituitary tumor. Pneumoencephalography showed no evidence of a solid tumor but a large thin-walled cystic area was found extending above the diaphragm of the sella turcica. (Fig. 1, A-E).

The patient was treated for the vertebral fracture and was discharged to return periodically for follow-up roentgenographic study of the sella turcica.

**CASE II.** A 44 year old white housewife had routine sinus roentgenograms for headache. The sinuses were not remarkable but the sella

turcica was enlarged and appeared eroded so that she was admitted for evaluation of suspected pituitary adenoma. The patient had been generally well except for labile hypertension and no physical abnormalities were noted other than mild hypertension. Endocrine studies including thyroid, adrenal and pituitary function were normal. Visual fields and spinal fluid were normal. Bilateral carotid angiograms were not remarkable. Pneumoencephalography, on early films, suggested extrasellar extension of a pituitary tumor; however, subsequently this was shown to be a cyst with very thin and smooth walls (Fig. 2, A-E).

The patient was discharged to return periodically for follow-up roentgenograms of the sella turcica.

### DISCUSSION

The older literature on arachnoid cysts is confusing since cysts of all types were grouped together. The leptomeningeal cysts are a distinct entity and are not considered here, although they have been reported to erode the base of the skull<sup>8</sup> and could conceivably involve the sella turcica. Starkman *et al.*,<sup>9</sup> in 1958, differentiated the primary or congenital intracranial arachnoid cysts that are continuous with normal arachnoid from secondary cysts developing after hemorrhage, trauma or infection. The primary cysts may occur at any age and Lewis,<sup>3</sup> in 1962, reported hydrocephalus in infancy due to arachnoid cysts. More recent reports of primary arachnoid cysts were published by Weinman<sup>12</sup> and Kruffy<sup>2</sup> in 1965. A review of the literature was given by all these authors.

Arachnoid cysts usually occur over the cerebral hemispheres in the sylvian fissure or posterior fossa. They are benign in themselves but may cause symptoms from pressure, depending on their location. Although

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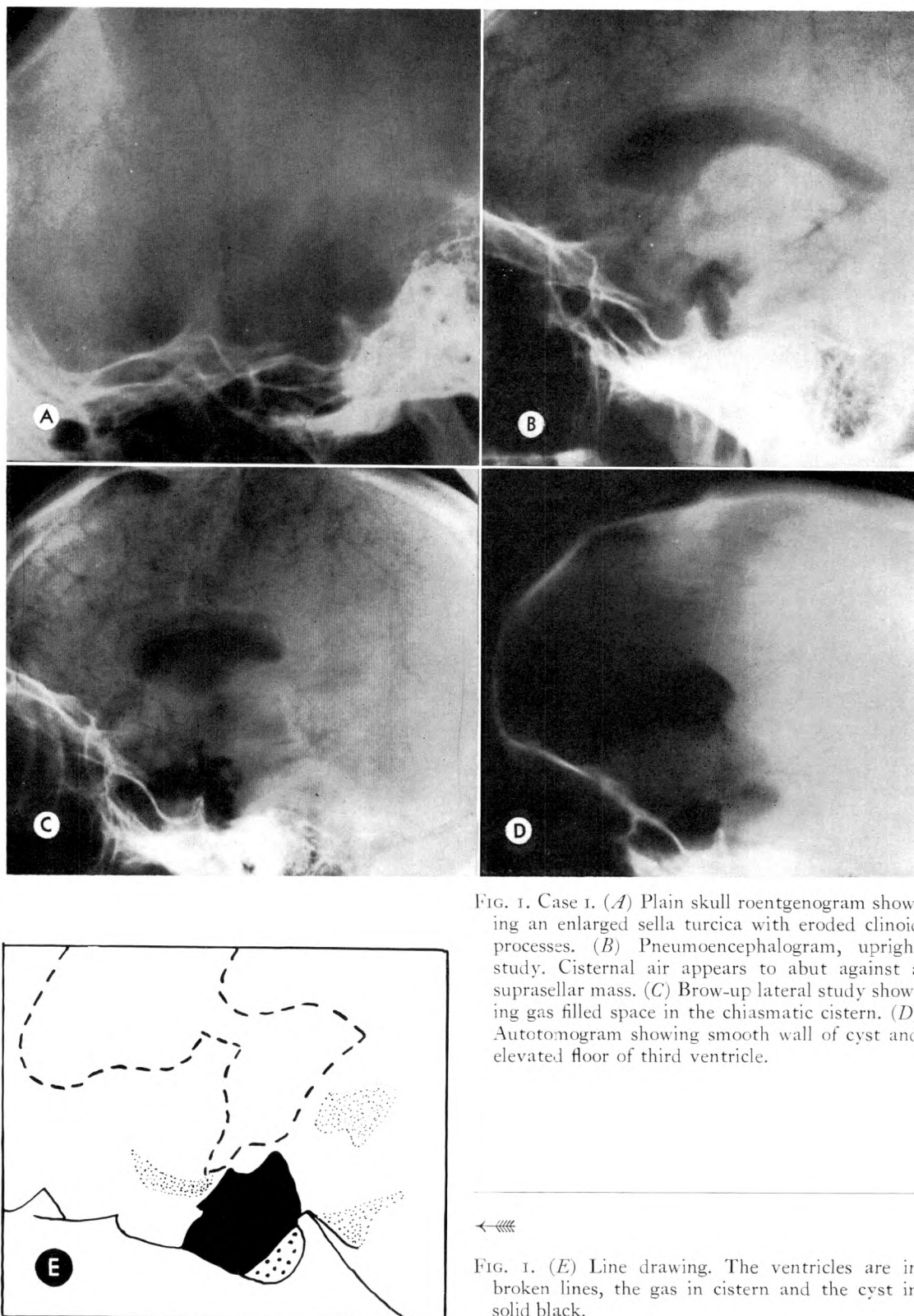


FIG. 1. Case 1. (A) Plain skull roentgenogram showing an enlarged sella turcica with eroded clinoid processes. (B) Pneumoencephalogram, upright study. Cisternal air appears to abut against a suprasellar mass. (C) Brow-up lateral study showing gas filled space in the chiasmatic cistern. (D) Autotomogram showing smooth wall of cyst and elevated floor of third ventricle.



FIG. 1. (E) Line drawing. The ventricles are in broken lines, the gas in cistern and the cyst in solid black.

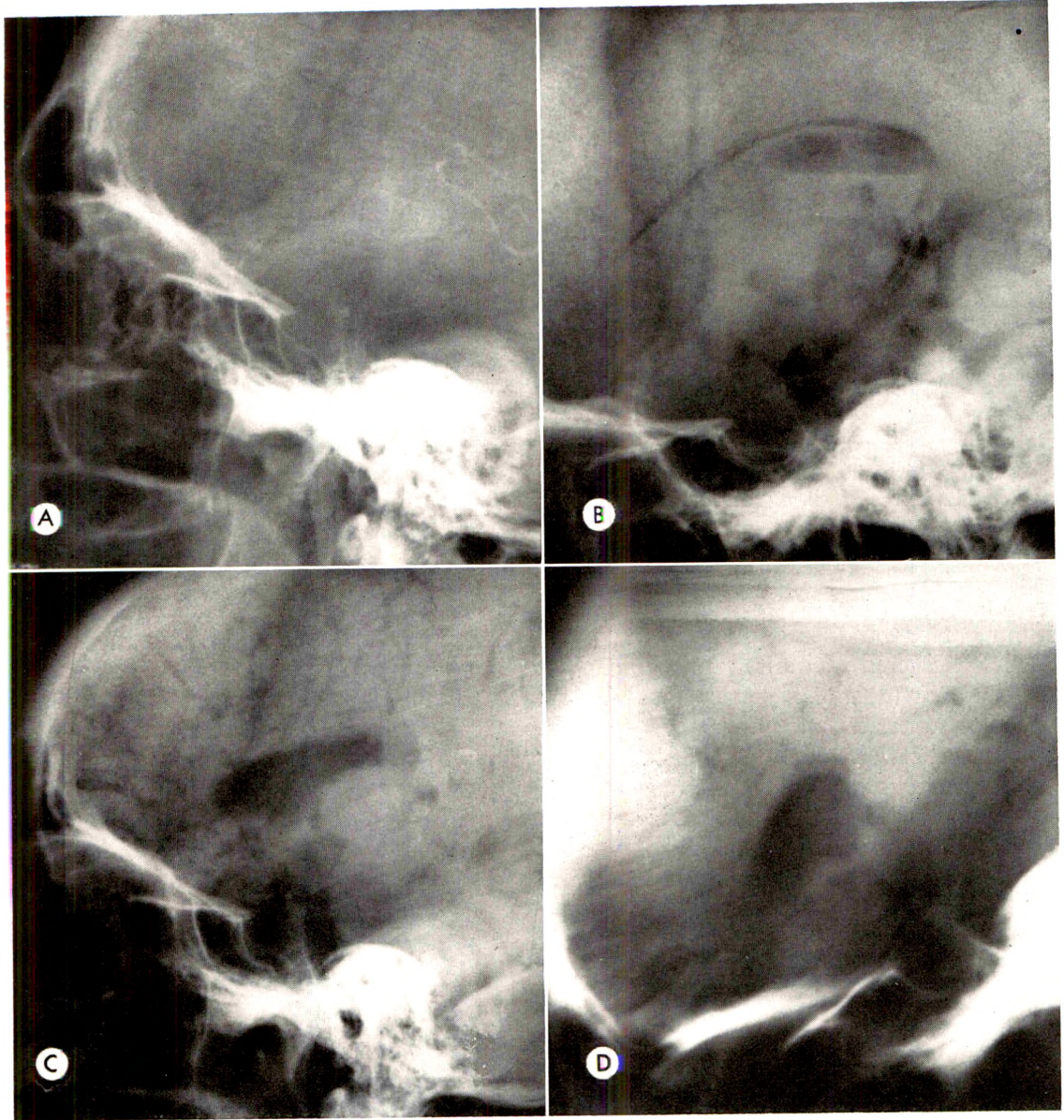


FIG. 2. Case II. (A) Lateral carotid angiogram showing central veins and large sella turcica. (B) Pneumoencephalogram, upright study. Gas in the chiasmatic cistern appears to be passing over a suprasellar mass. (C) Brow-up lateral study shows gas filled space in the chiasmatic cistern. (D) Autotomogram showing elevated floor of the third ventricle and no solid tumor.

over a hundred cases have been reported in the literature, only 2 cases of arachnoid cysts eroding the sella turcica could be found. The first of these was described by Twining *et al.*<sup>11</sup> in 1936 in a discussion of roentgenographic changes with pituitary tumors. Robertson,<sup>5</sup> in his text on pneumoencephalography, presents 1 case and de-

scribes the findings without comment. There is a striking similarity in the roentgenographic appearance of the sella turcica in these cases and our Case I. Both Twining and co-workers' and Robertson's cases had surgery and in each case only a thin walled cyst of normal arachnoid tissue was found.



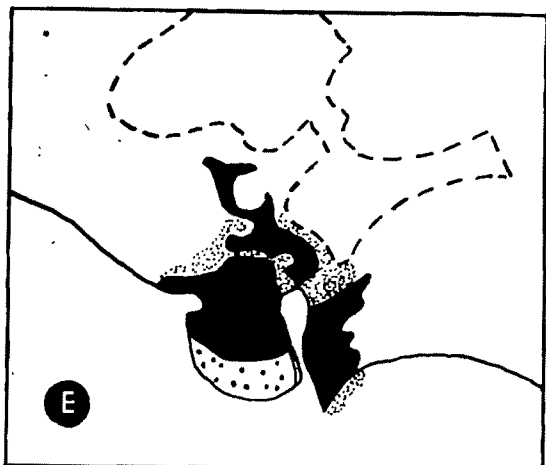


FIG. 2. (E) Line drawing. The ventricles are in broken lines, the gas in cistern and cyst in solid black.

As surgery was not performed in either of our cases, the diagnosis is presumptive, but is considered warranted for the following reasons: (1) the absence of solid elements of tumor on pneumography; (2) the similarity between our cases and those reported previously; and (3) the circumstance of having a 5 year follow-up in Case 1 without evidence of further sella change which would be highly unlikely in a cystic tumor.

These cysts may arise in the small subarachnoid space described by Di Chiro<sup>1</sup> and by Taveras and Wood<sup>10</sup> between the diaphragma sellae and the pituitary. Pneumoencephalography is diagnostic due to the fact that cystic tumors in this area always have some solid elements that are recognizable with careful study. Since air lying laterally in the sylvian fissure or in the cisternal space about the carotids, as described by Lewtas and Jefferson,<sup>4</sup> may be confusing, it is imperative that the midline structures be seen. This is best accomplished by autotomography in the hanging head position as described by Schvarcz<sup>7</sup> in 1959 and Schechter and Jing<sup>6</sup> in 1960, in which the floor of the third ventricle is seen as well as the intra- and suprasellar areas.

This condition is probably not as rare as the paucity of reports in the literature would indicate. Several neuroradiologists

who were questioned had seen or heard of a similar case and the absence of reports is probably due to reluctance in reporting an isolated case. This condition should be included in the differential diagnosis of sella turcica erosions, especially when these occur in the absence of ocular or hormonal changes. Once the cysts are filled with air, they are presumably in free communication with the subarachnoid space and should cause no further damage. The importance of correct diagnosis is in avoiding unnecessary therapy and points out the fallacy of treating lesions in the sella turcica area without complete roentgenologic evaluation.

#### SUMMARY

Two cases of primary arachnoid cysts in the pituitary fossa are reported. This is a rare condition but should be included in the differential diagnosis of processes causing erosion of the sella turcica. The diagnosis may be established by pneumoencephalography.

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## EXTRASELLAR EXTENSION OF PITUITARY ADENOMAS

### CLINICAL AND NEURORADIOLOGICAL CONSIDERATIONS

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THE treatment of pituitary tumors requires an early and exact assessment of the extent of the neoplasm for the purpose of adequate therapy not only by the neurosurgeon but by the radiotherapist as well. In addition, close cooperation between the neurological surgeon, neuroophthalmologist, endocrinologist and diagnostic and therapeutic radiologist is imperative for the successful management of patients suspected of harboring a sellar or parasellar mass lesion. The neurological, endocrinological and plain skull roentgenographic findings, important as they are, allow in most instances no definite clue as to the etiology of the lesion,<sup>27</sup> unless there is evidence of a hypersecreting tumor such as an eosinophilic adenoma or hyperostosis of the tuberculum sellae commonly associated with meningioma at this site.

Clinical and roentgenologic findings may occasionally but not consistently suggest the presence of a large mass. However, these are not sufficient to delineate the exact extent of the tumor, nor do they offer any reliable information as to the relationship of the tumor to the adjacent neural and vascular structures.

Extensive extrasellar growth is more often observed in chromophobe adenomas. However, Cushing and Davidoff<sup>6</sup> described 4 cases of acromegaly with large extrasellar extension. Erosion of the sella is most unusual in basophile adenoma, although occasionally a case is encountered in which a basophile adenoma enlarges the sella to such an extent that pressure phenomena necessitate surgical intervention.

As White and Warren<sup>30</sup> pointed out,

little attention has been paid to the potential danger of letting a pituitary neoplasm grow to an unusual size. This is particularly apt to occur with chromophobe adenomas in instances of unusual fixation of the chiasm with absent or only late involvement of the decussating fibers in the optic chiasm. However, it is not unusual to find also examples of very large chromophobe or mixed pituitary adenomas associated with visual impairment, which were allowed to attain unusual size as the result of either procrastination, prolonged or overenthusiastic use of radiotherapy, failure of early diagnosis and omission of early use of pneumography and/or angiography. In these patients, the neoplasms may extend into unusual areas, causing a departure from the classic clinical syndrome, rendering the diagnosis difficult and the surgical treatment prohibitively hazardous. Also the prospects of a satisfactory response to radiotherapy are not very bright in such widely extending adenomas,<sup>14</sup> the exact size of which is not even known or suspected. Although it is frequently assumed that unusual degrees of extrasellar extension are rare, it occurred at a rate of 22 per cent in 338 patients of the Cushing series<sup>15</sup> and in 14 per cent of Jefferson's<sup>17</sup> 128 cases.

Cerebral pneumography and angiography are the only methods which permit an accurate delineation of intracranial extrasellar extension of a pituitary neoplasm and should therefore be carried out before any irradiation or surgical treatment is contemplated. Radioisotope scanning has likewise been used for the localization of suprasellar masses. However, due to the fact that

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the isotope concentration is as high in the temporal muscle as in the tumor, only large neoplasms extending well above the sella turcica can thus be detected.<sup>27</sup> The method, although useful, is therefore only of limited value for the localization of tumors of the hypophyseal and suprasellar region. Although the significance of these diagnostic procedures has been more widely recognized in recent years by neurological surgeons and neuroradiologists as well, they have often been considered superfluous or their importance has not been sufficiently appreciated by the general radiologist, radiotherapist or internist who are often called upon to treat pituitary neoplasms.

Pneumography in most instances, even of limited suprasellar tumor extension, will delineate a pituitary tumor by evidence of encroachment on the basal cisterns and the infundibular portion of the third ventricle. Cerebral angiography, which also allows an estimate of the tumor size, particularly in larger neoplasms, may furnish additional valuable information concerning the relationship of the tumor to the arteries of the circle of Willis and to the cavernous portion of the internal carotid artery. Furthermore, displacement of the internal cerebral and basilar veins can be detected in tumors of larger size, particularly with predominantly superoposterior direction of growth. In addition, angiography permits, in most instances, to exclude the presence of an arterial aneurysm which worked its way into the sellar and suprasellar area, simulating a pituitary tumor clinically as well as roentgenographically. White and Ballentine<sup>29</sup> collected and described 36 cases of intrasellar aneurysms simulating hypophyseal tumors, mentioning briefly 12 other instances. They implied that routine angiography should be employed in hypophyseal tumor suspects. Occasionally, the angiogram will also reveal the etiology of a chiasmal and hypopituitary syndrome by demonstrating increased vascularity or a tumor stain, as in the case of a tuberculum sellae meningioma.<sup>2,4,27</sup>

It is the intention of the authors to illus-

trate and to re-emphasize the relative value of pneumography and angiography in cases of pituitary neoplasms with extrasellar extension and to furnish some additional information for a fuller understanding of the subject.

#### CLINICAL AND DIFFERENTIAL DIAGNOSTIC CONSIDERATIONS

The neuroradiological diagnosis of expanding pituitary adenomas requires not only familiarity with the normal and pathological roentgenological as well as topographical anatomy of the sella turcica and its adjacent structures, but also knowledge of the differential diagnostic features of other lesions in the sellar or juxtasellar area and, in addition, some information concerning the clinical and pathological manifestations of pituitary tumor spread. This information, available in textbooks, monographs and numerous publications of the neurological, neurosurgical and neuroradiological literature, can obviously not be discussed completely or in detail. However, some aspects, necessary for a fuller understanding of the various phenomena of extrasellar extension of pituitary tumors and the problems of their differential diagnosis, will be mentioned.

Extrasellar growth of hypophyseal adenomas is unpredictable and, according to Jefferson,<sup>17</sup> dependent upon several factors: the growth urge of the adenoma, the state of fixation of the chiasm, the shape of the pituitary fossa and the nature of its diaphragm. The tumor can predominantly grow in one direction or in various directions simultaneously. Erosion of the confines of the sella in posterior, inferior and anterior directions is the earliest and most frequent manifestation of extension. In most instances the initial roentgenologic diagnosis of an intrasellar mass depends upon the enlarged size and abnormal shape of the sella turcica, although chromophobe pituitary tumors with even large suprasellar extensions may occasionally be encountered in the presence of a normal sella turcica,<sup>2,7,24</sup> or a small intrasellar mass may be con-



nected by a narrow neck with large bulbous extensions into the frontal or temporal lobes.<sup>27</sup> Of course, a ballooned-out sella turcica does not indicate the presence of a pituitary adenoma. It can occur with craniopharyngiomas, in extension of a tuberculum sellae meningioma or arterial aneurysm into the sella, with inflammatory involvement of the hypophysis, with marked dilatation of the floor of the third ventricle or increased intracranial pressure, and also metastatic tumors of the pituitary as well as malignant tumors of the sphenoid bone (chordoma, myeloma, sarcoma) may simulate extensive pressure erosions of the sella turcica as seen with large pituitary tumors.<sup>25,27,30</sup>

The presence of intra- or suprasellar calcifications is also of limited diagnostic value in the diagnosis of the type or extent of a sellar tumor.<sup>25,27</sup> Intracellular calcifications occur in 5 to 10 per cent of pituitary adenomas; they may vary from fine speckling to a dense mass.<sup>27</sup> Suprasellar calcifications are found only infrequently in pituitary adenomas<sup>24</sup> and in 50 per cent or 70 to 80 per cent<sup>25,27</sup> of the craniopharyngiomas. Similar calcifications may be less frequently seen in meningiomas, dermoid tumors or aneurysms.<sup>27</sup> The ringed or eggshell type of calcification is apt to occur in aneurysms, craniopharyngiomatous cysts or dermoid tumors.<sup>27,29</sup> Rand<sup>24</sup> observed a ring-like calcification outlining the marked extrasellar extent of a chromophobe pituitary adenoma, and, also, Davidoff and Epstein<sup>7</sup> observed a curvilinear streak of calcification in an adenoma. White and Ballentine<sup>29</sup> cited 4 cases in which suprasellar calcifications in an aneurysm were misinterpreted, leading to subsequent exploration for suspected craniopharyngiomas.

Aneurysms which produce compression of the optic nerves and chiasm<sup>4,16,27</sup> or simulate pituitary tumors<sup>2,29</sup> originate most often from the internal carotid artery, although some project downward from the anterior cerebral or anterior communicating arteries.<sup>27,29</sup> In one reported instance, a

huge sac protruded forward into the sella turcica from the rostral end of the basilar artery.<sup>16</sup> Chase and Taveras<sup>4</sup> found that the aneurysms usually arose from the internal carotid artery just distal to or at the origin of the ophthalmic artery or from the anteromedial aspect of the supraclinoid portion. White and Ballentine<sup>29</sup> felt that the majority of aneurysms simulating pituitary tumors probably originated from the infraclinoid segment.

Roentgenograms of the skull showing ipsilateral erosion of the anterior clinoid process or widening of the supraorbital fissure may arouse suspicion of an aneurysm. Erosion of the lower and outer wall of the optic canal or union of the optic canal with the supraorbital fissure has been regarded as pathognomonic or almost pathognomonic of an aneurysm.<sup>16</sup> However, other authors believe that similar osseous changes may be seen in tumors producing enlargement of the sella turcica or in pituitary chromophobe adenomas.<sup>7</sup>

#### PATHWAYS OF TUMOR EXTENSION

Several pathways of extrasellar extension of pituitary adenomas have been described by various authors on the basis of autopsy and operative findings.<sup>8,15,17,30</sup> Jefferson<sup>17</sup> listed a frontal, temporal, hypothalamic, posterior and pharyngeal type. White and Warren<sup>30</sup> distinguished 6 possible pathways: pharyngeal, hypothalamic, frontal, temporal extension, invasion of the cavernous sinus, and posterior subtentorial extension.

1. *Extension into Sphenoid Sinus and Nasopharynx.* Erosion of the sellar floor with extension of the tumor into the sphenoid sinus is the most common type. As mentioned above, invasion of the sphenoid sinus can be easily recognized in many cases on skull roentgenograms.<sup>24</sup> However, this event may remain clinically unrecognized and may actually delay the onset of the classic symptoms unless, in rare instances, rhinorrhea should supervene, or unless the tumor penetrates into the nasopharynx.<sup>2,4,30</sup> This may manifest itself by

increasing nasal discharge and obstruction. A mass in the roof of the nasopharynx can often be demonstrated. There were 8 examples of this in Cushing's series as reported by Henderson.<sup>15</sup>

2. *Hypothalamic Extension.* Compression and displacement of the hypothalamus occurs if the tumor extends upward and backwards usually behind a prefixed chiasm. In spite of considerable involvement of the hypothalamus, there may be a paucity of symptoms aside from headaches, visual impairment, apathy and stupor. Hypothalamic symptoms such as polydipsia and polyuria or cardiac, respiratory or temperature regulatory disturbances are uncommon unless actual destruction or invasion of hypothalamic structures occurs.<sup>17,27,30</sup> If the tumor reaches the foramen of Monro and thus blocks the cerebrospinal fluid outflow, severe headache, papilledema and hydrocephalus may occur.<sup>7</sup> Some of the mentioned features including polydipsia were observed in 1 of our patients (Case III, Fig. 5, A and B).

3. *Frontal Extensions.* If a tumor grows in a forward and upward direction emerging, as it were, in front of a post-fixed chiasm, it may extend into the anterior fossa, compressing the olfactory bulbs, separating and compressing the frontal lobes or invading one or both frontal lobes. These frontal extensions are usually larger than the intrasellar components<sup>17</sup> and they may be either solid or cystic in character. White and Warren<sup>30</sup> reported in detail 1 patient with combined frontal and posterior subtentorial growth. In a review of the literature, they found only 4 examples of frontal extension on record. They commented briefly on a similar case encountered by one of the authors. Personality changes, epileptic seizures, olfactory and visual disturbances are commonly encountered in these patients. We observed 1 patient with a solid intrasellar chromophobe adenoma and a cystic component extending into the right frontal lobe. The symptoms and findings in this patient consisted of headache,

lack of concentration, unilateral superior-temporal quadrantanopia, diplopia and papilledema (Case v).

4. *Temporal Extension.* Adenomas extending in a lateral direction between the cavernous sinus and the chiasm invade the middle cranial fossa, impinging upon the medial aspect of the temporal lobe and the optic tract, causing temporal lobe seizures with olfactory aura and homonymous hemianopia.<sup>15,17,21,27,30</sup> Davidoff and Epstein<sup>7</sup> demonstrated pneumoencephalographic evidence of a large eosinophilic pituitary adenoma extending out of a relatively normal sella turcica into the temporal fossa.

5. *Involvement of the Cavernous Sinus.* The cavernous sinus may be affected by intrinsic pressure on its medial wall by an expanding tumor. Actual invasion of the cavernous sinus and Meckel's cave occurs when the adenomas have taken on malignant characteristics.<sup>17</sup> Weinberger, Adler and Grant<sup>28</sup> described 14 cases with cavernous sinus invasion in a series of 169 cases of verified pituitary adenomas. Extraocular palsies, irritation of the ophthalmic division of the trigeminus or trigeminal anesthesia and engorgement of orbital veins are the resultant manifestations of cavernous sinus invasion.<sup>17,30</sup> Mere displacement of the cavernous sinus and distortion of neural structures may be responsible for incomplete extraocular palsies. In 3 of our 6 patients who had either diplopia, or in 1 instance ptosis, the symptoms disappeared following surgical intervention. In a previously reported case of massive hemorrhage into a presumably chromophobe adenoma, a spontaneous total recovery of bilateral external ophthalmoplegia, severe visual impairment and hypopituitarism occurred, indicating that the extraocular nerves can be severely affected by extrinsic pressure on the cavernous sinus alone.<sup>18</sup>

6. *Posterior Subtentorial Extension.* The posterior and inferior extension of pituitary adenomas is the most unusual form. White and Warren<sup>30</sup> reported a case of this type





FIG. 1. Normal lateral brow-up pneumogram, showing basilar cisterns traversed by optic nerves and well-filled infundibular portion of the third ventricle.

with simultaneous frontal expansion. Weinberger, Adler and Grant<sup>28</sup> described 2 cases with simultaneous tumor growth into the subtentorial space, in 1 instance presenting itself as a cerebellopontine angle tumor with erosion of the petrous apex, cerebellar signs and unilateral paralysis of the third, fifth, sixth and eighth cranial nerves as well as bilateral papilledema, with reduced vision but without visual field defects.

#### ELEMENTS OF PNEUMOGRAPHIC DIAGNOSIS

The demonstration of pituitary adenomas and the assessment of their size on the pneumogram (Fig. 1) depend on the degree of their suprasellar extension.<sup>1,3</sup> The resultant encroachment on the basal cisterns and indentation of the third ventricle or even of the frontal horns of the lateral ventricles are best seen on lateral exposures. Although the anteroposterior view reveals the filling defect of the infundibular portion of the third ventricle (Fig. 4 through 6) and in cases of unilateral suprasellar growth, a shift of the ventricular system (Fig. 7B), it usually fails to reveal the lateral extent of the tumor. Even when anteroposterior projections with central ray angulation of 20 to 35 degrees are used in order to better

demonstrate the suprasellar cisterns and olfactory sulci or the encroachment upon them, the lateral extent of the tumor is often not demonstrated.<sup>2</sup> Of course, large infratemporal extensions are known to cause elevation of the involved temporal horn.

Bakay and White<sup>3</sup> introduced a classification based upon the degree of suprasellar extension of pituitary adenomas. An extent of 0.5 cm. already caused an intrusion into the chiasmatic and interpeduncular cisterns and with an extent to 1 cm. the inferior portion of the third ventricle was reached. Extension of 2 and 3 cm. produced definite to marked indentations of the third ventricle. Further extent to 3.5 cm. was consistent with encroachment on the foramen of Monro, although this was rarely obstructed. Also, indentation and elevation of the anterior horns of the lateral ventricles occurred at this degree of suprasellar extension. Similar degrees of suprasellar growth were noted in our patients as shown in Figures 3 through 6. The tumors usually project upwards and caudally as a cone or cone-shaped mass, wedging themselves between the chiasmatic and interpeduncular cisterns. Although some authors<sup>6,7</sup> termed the obliteration of the cisterna chiasmatis in pituitary adenomas as characteristic, this was not always observed in our material of pituitary adenomas, nor had this been the experience of other observers.<sup>2,3,17</sup> However, obliteration of the chiasmatic cistern was also noted in 1 of our cases of intrasellar craniopharyngioma with considerable suprasellar extent. This patient had been treated under the diagnosis of chromophobe adenoma and had received two courses of radiation therapy in two other hospitals during recent years. Since contrast studies had not been carried out, the actual size of the tumor also remained unknown.

#### ELEMENTS OF ANGIOGRAPHIC DIAGNOSIS

The angiographic diagnosis of pituitary adenomas which grow exclusively within the enlarged sella turcica is primarily de-

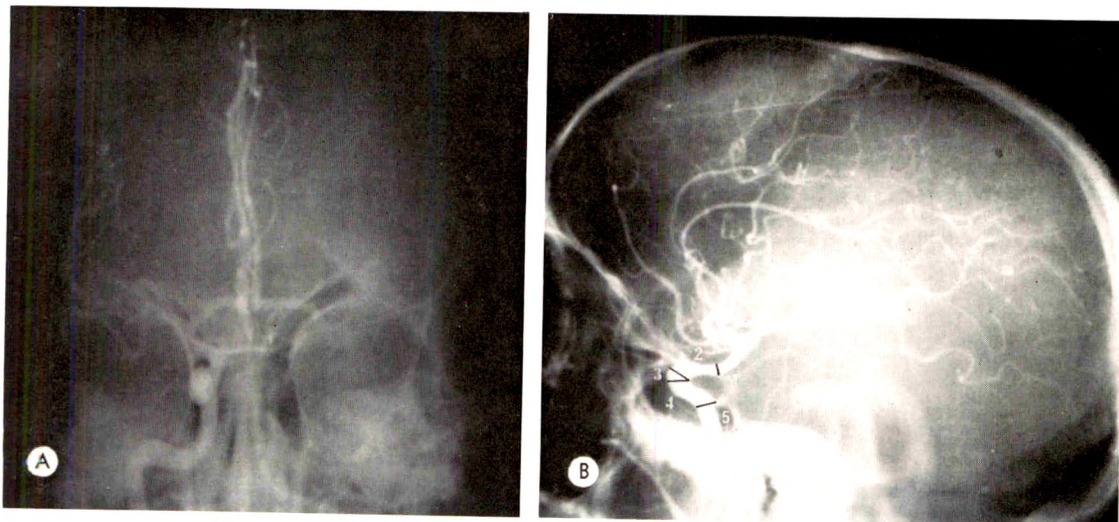


FIG. 2. (A) Normal right angiogram. (B) Normal left angiogram (numbers indicate the various segments of the carotid siphon according to Fischer (as cited by Ecker and Riemenschneider<sup>10</sup>). (1) Supraclinoid segment; (2) infraclinoid (intradural) segment; (3 and 4) anterior and posterior portion of intracavernous segment; and (5) precavernous segment.

pendent upon displacement of parts of the intracavernous segment of the carotid artery (Fig. 2B). Suprasellar extension of the adenomas, at least of appreciable degree, manifests itself mainly by alterations of the supraclinoid segment, the carotid bifurcation, the anterior cerebral arteries, and, to a lesser degree, by deflections of the anterior choroidal, posterior commu-

nicating, posterior cerebral arteries, and, rarely, of the pericallosal arteries. With tumor growth in predominantly superoposterior and lateral directions, displacement of the basilar vein and/or of the internal cerebral veins is encountered (Fig. 10).

Due to the frequency of normal variations and asymmetry in the course of the

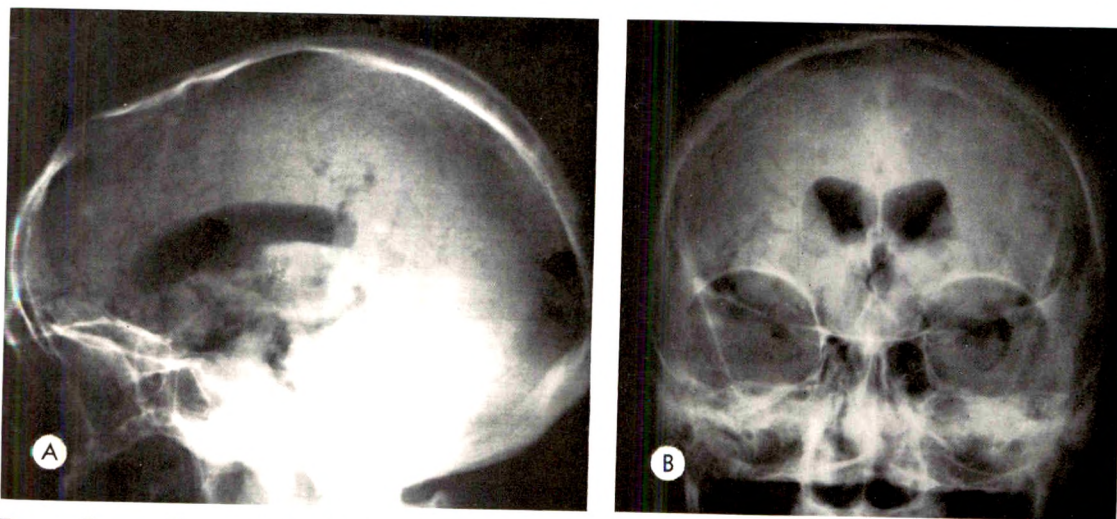


FIG. 3. Case 1. Chromophobe pituitary adenoma in a 53 year old male. (A) Lateral pneumogram shows moderately-sized posterosuperior extension of adenoma, wedged between the well-filled chiasmatic and interpeduncular cistern. Note encroachment on 3rd ventricle. (B) Anteroposterior view demonstrates partial obliteration of infundibular portion of 3rd ventricle.



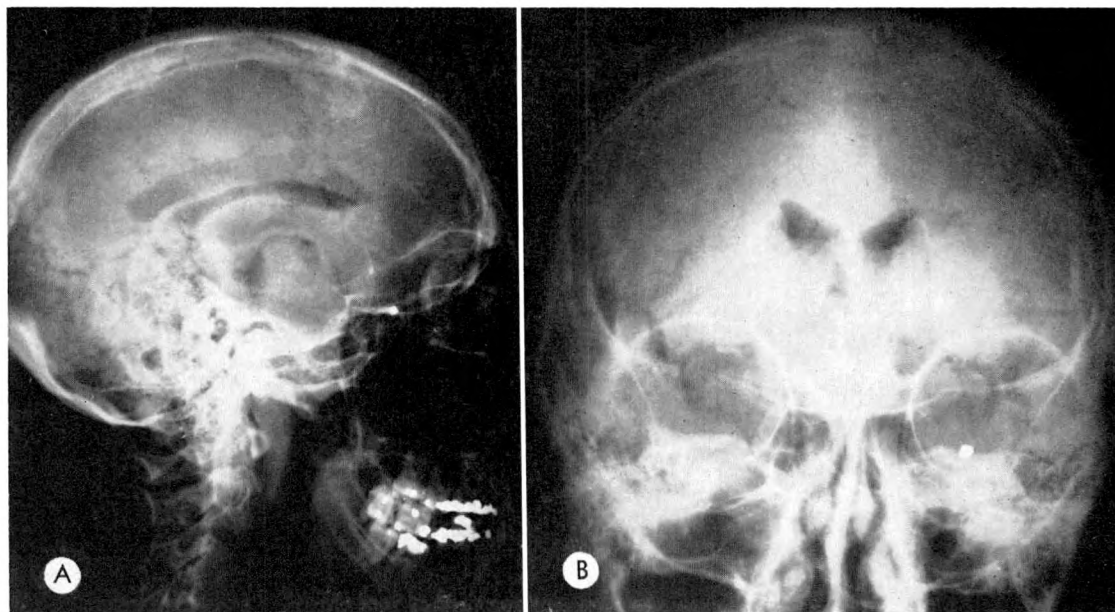


FIG. 4. Case II. Chromophobe pituitary adenoma, partially cystic, in a 35 year old male. (A) Lateral pneumogram shows a fairly large, predominantly upward, suprasellar extension. Note distortion and sparse filling of chiasmatic and interpeduncular cistern. (B) Anteroposterior exposure shows obliteration of infundibular portion with slight displacement of 3rd ventricle to the right.

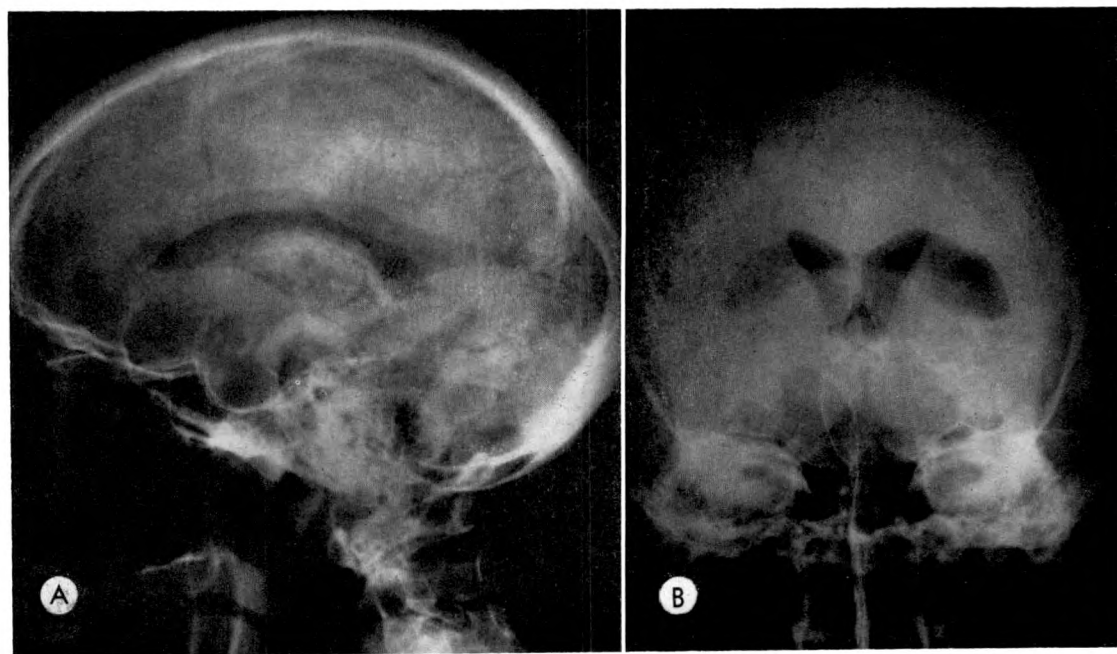


FIG. 5. Case III. Chromophobe pituitary adenoma with marked hemorrhage (25 cc.) into the tumor in a 35 year old acromegalic male. (A) Lateral pneumogram shows an enlarged sella turcica and a large suprasellar mass obliterating the anterior and inferior portion of the 3rd ventricle, the cisterna chiasmatis and partially the cisterna interpeduncularis. (B) Posteroanterior view shows obliteration of the lower half of the 3rd ventricle.

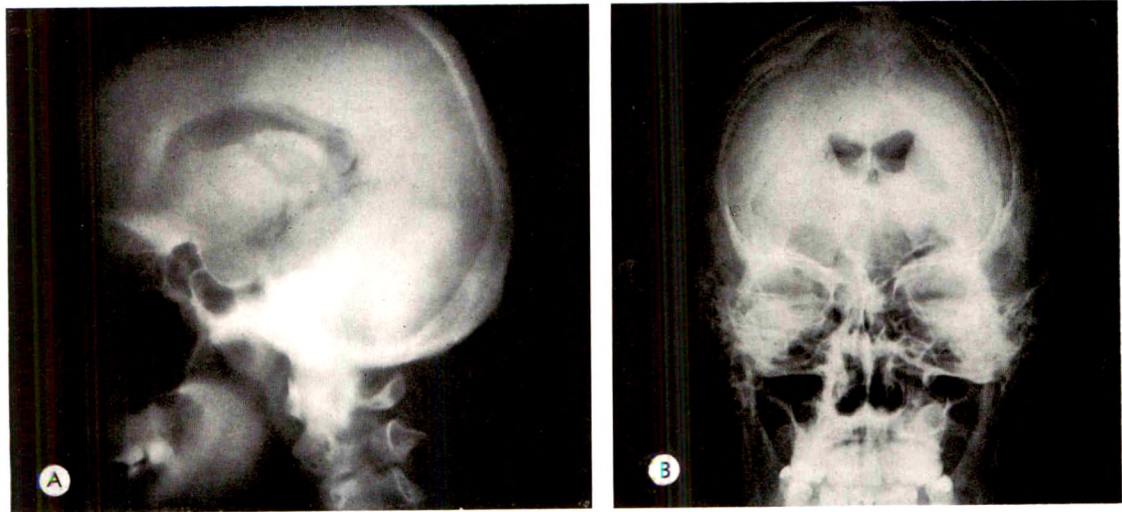


FIG. 6. Case iv. Chromophobe pituitary adenoma in a 26 year old male schizophrenic. (A) Midline lamina-gram of pneumogram demonstrates an enlarged sella turcica and a suprasellar mass with predominantly superior extent, obliterating the basilar cisterns, the anterior and infundibular portion of the 3rd ventricle and encroaching on the frontal horn and foramen of Monro. (B) Anteroposterior pneumogram shows obliteration of the lower portion of the 3rd ventricle and encroachment on the left lateral ventricle.

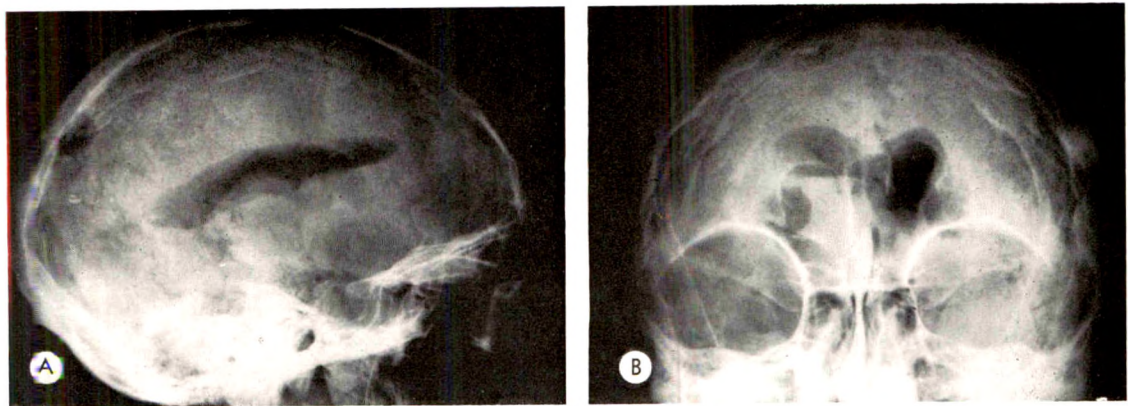


FIG. 7. Case v. Chromophobe pituitary adenoma in a 24 year old male. Solid tumor in enlarged sella turcica with large cystic component (35 cc.) in right frontal lobe. (A) Left lateral ventriculogram demonstrates encroachment of a large frontal mass on the right lateral ventricle with elevation and thinning of the frontal horn and anterior third of the ventricular body. A mass is also seen protruding from an enlarged sella turcica, wedging its dome between the chiasmatic and interpeduncular cisterns. (B) Anteroposterior view shows elevation and distortion of the right lateral ventricle and shift of the ventricular system to the left. (A shift and bowing of the right pericallosal artery had been noted on a previous angiogram.)

anterior cerebral arteries (Fig. 2, A and B) and supraclinoid segments of the carotid arteries, small deflections, even if asymmetrical, may have little clinical significance.<sup>27</sup> Therefore, normal variations should be kept in mind to avoid too many pathological diagnoses.

Some authors described asymmetrical

displacements of the carotid siphon as characteristic for all sellar and suprasellar growths (Fig. 8, E and F; and 9, C and D). Bilateral angiograms are, therefore, necessary in order to arrive at a proper evaluation.<sup>2,11</sup> A clinically significant displacement occurs, however, only when a lesion has attained a certain critical size. Accord-



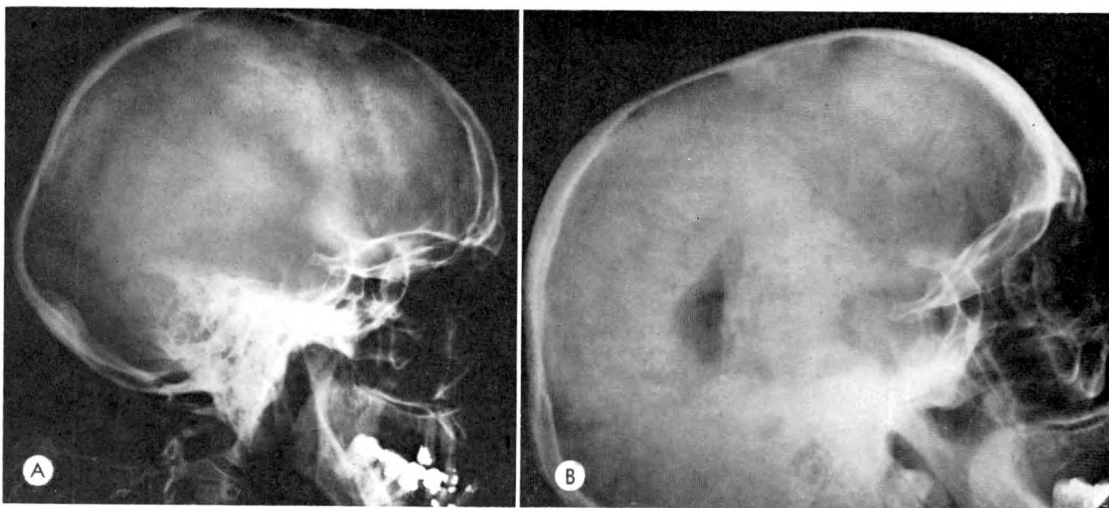


FIG. 8. Case VI. Chromophobe, mainly solid, pituitary adenoma in a 44 year old male. (A) Roentgenogram reveals enlarged sella turcica with thinning of dorsum sellae and posterior clinoids. (B) Lateral pneumogram shows a posterosuperior extension of a moderate-sized tumor between the chiasmatic and interpeduncular cistern.

ingly, pituitary tumors, even with considerable dorsal protrusion or posterior extension, may be present without disturbing the anterior cerebral arteries or the T of the internal carotid artery<sup>26,27</sup> (Fig. 8, *E* and *F*). It is for this reason that several groups of workers consider pneumography more reliable in the visualization of pituitary adenomas with smaller or moderate superior extension or even larger posterior extension, although it is conceded that tumors extending high above the sella turcica may be well delineated by either method.<sup>2,27</sup> In the determination of lateral extension of the tumor, angiography appears superior.<sup>2,11</sup>

#### DISCUSSION

The value of neuroradiological contrast studies in the diagnosis and management of pituitary tumors or other neoplasms of the sellar or parasellar region has been more generally appreciated only in recent years. As late as 1955, Davidoff and Epstein<sup>7</sup> explained the relative paucity of contributions dealing primarily with the pneumoencephalographic aspects of pituitary adenomas: "The obvious reason is that the diagnosis can usually be made without such

aid." However, already in 1940 Jefferson<sup>17</sup> strongly recommended the use of pneumoencephalography, indicating that the suprasellar extent of a pituitary adenoma is best assessed by this method.

A radical change in the attitude of many neurosurgeons and neuroradiologists towards the use of either pneumography and/or angiography emerged in the past decade. To judge by the original articles published on this subject since 1950, there is unanimity of opinion that a contrast study should be performed in all pituitary adenomas or other chiasmal lesions before radiation or surgical intervention is contemplated. However, the question whether pneumography or angiography is the preferable procedure or whether both should be employed, and in what sequence, has remained a controversial subject. Several authors<sup>20,23,26,27</sup> accept pneumography as the method of choice in patients suspected of harboring a lesion within the sellar or juxtaseellar area. With rising popularity and diagnostic refinement of cerebral angiography, this opinion has been challenged by some authors. Lima<sup>19</sup> stated: "The principal contribution of cerebral angiography in the management of hypophyseal ade-

nomas resides in the fact that it is capable, with greater precision and greater security than a simple clinical and radiological examination and ventriculography, of guiding the decision of the physician, the technique of the neurosurgeon, and the mode of action of the radiotherapist." Also Driesen and Schmidt<sup>9</sup> asserted that carotid angiog-

raphy is the method of choice for the investigation of sellar lesions. On the other hand, Lindgren<sup>20</sup> and Walker<sup>27</sup> reached the conclusion that pneumography gives a greater accuracy than any other single technical method of examination in chiasmal syndromes. Udvarhelyi *et al.*<sup>26</sup> state that 76 per cent of 25 patients with suprasellar

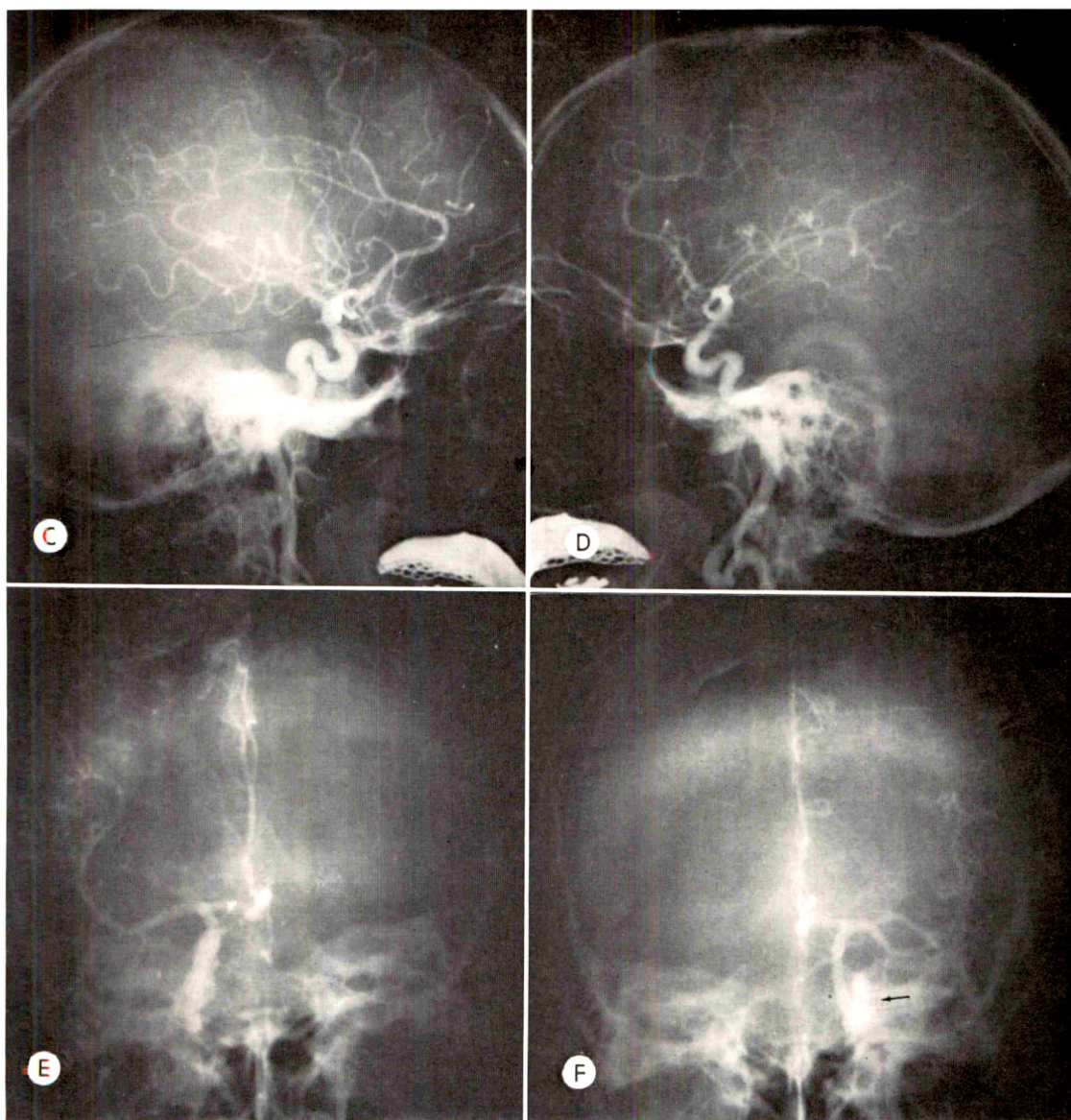


FIG. 8. (C) Normal right lateral angiogram. (D) Left lateral angiogram shows opening of the siphon with elevation and straightening of supraclinoid carotid segment. (E) Normal right anteroposterior angiogram. Note superimposition of anterior and posterior segments of the carotid artery. (F) Left anteroposterior angiogram reveals lateral displacement of posterior segment of intracavernous portion of the left carotid artery (arrow).



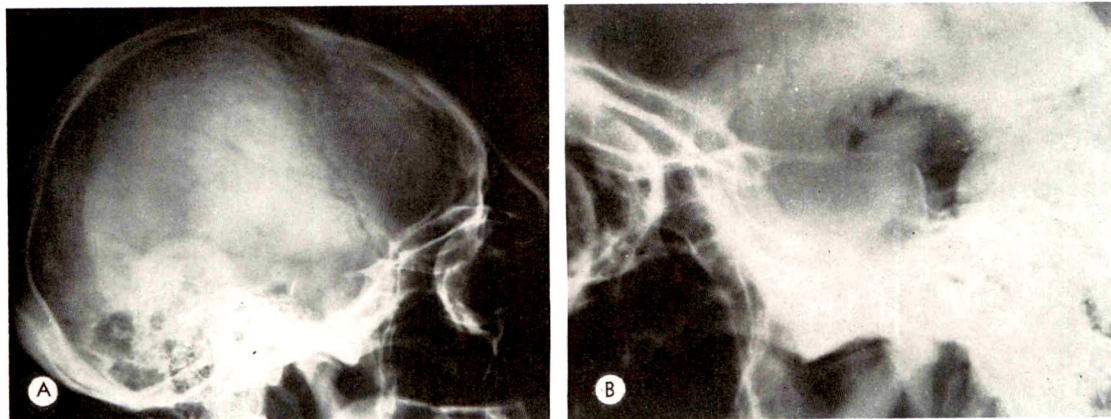


FIG. 9. Case VII. Chromophobe pituitary adenoma, mainly cystic, in 45 year old male acromegalic. (A) Lateral skull roentgenogram in 1956 showed enlarged sella turcica with erosion of the dorsum sellae, posterior clinoid processes and of the sellar floor extending to the ethmoid area. (B) Lateral pneumogram in 1956 showed a finger-like superoposterior extension wedged between the chiasmatic and interpeduncular cisterns with encroachment on the infundibular portion of the 3rd ventricle.

masses had positive pneumoencephalograms. Only 34 per cent had positive angiographic findings. In 57 per cent of craniopharyngiomas, 90 per cent of pituitary adenomas and 71 per cent of suprasellar meningiomas, the pneumoencephalographic findings were positive. Poppen<sup>23</sup> had previously expressed the opinion that the pneumoencephalogram gave a more accurate and definite localization of the tumor and, additionally, an approximate localization of the optic chiasm.

Several authors<sup>2,4,10,12</sup> feel that pneumography and angiography have different potentialities and should, therefore, be used as complementary procedures. Fager<sup>12</sup> employs both methods in all intrasellar tumors before any form of treatment is instituted. Subsequent clinical or radiological evidence of extension of the adenomas following radiotherapy serves as indications for surgical intervention. Chase and Taveras,<sup>4</sup> in a comparative study of both methods, reached the conclusion that "pneumography is superior to angiography in that it gives an accurate indication of the size and shape of the tumor, as well as its relationship to the third ventricle, the foramen of Monro, and the brainstem." Although they admit that small tumors of the tuberculum sellae are difficult to detect

on the angiogram and also that posterior suprasellar tumors without intrasellar components are particularly difficult to diagnose angiographically, they feel that careful angiographic evaluation permits one to arrive at a surprisingly accurate appreciation of the actual size of the tumor and its relationship to the sella turcica. Angiography, in addition, offers the obvious advantage of demonstrating an aneurysm or a tumor stain, thus allowing an etiologic diagnosis. Similar views are held by El-Banhawy and El-Nadi,<sup>11</sup> who concluded that "encephalography provides us with a cross-section of the tumor in the sagittal plane at the middle line, while angiography illustrates a cross-section in the coronal plane at the level of the anterior cerebral arteries." They recommend pneumography as the primary procedure. However, if surgical intervention is planned, angiography is added.

Bakay and Bean<sup>2</sup> express the opinion that arteriography may supplement but not supplant pneumography in cases of pituitary adenomas. In the diagnosis of suprasellar meningiomas, angiography proved to be more reliable, whereas air studies were the method of choice for large craniopharyngiomas. They mentioned as additional potential advantages of an-



giography, aside from those mentioned by other authors, its use in postoperative followup studies, since obliteration of the suprasellar cisterns, especially the chiasmatic cistern, may follow transfrontal exploration of the sella turcica, it may also be

of help in excluding a mass lesion in cases of optic chiasmatic arachnitis.<sup>2</sup>

Since the cisterna chiasmatis, according to Davidoff and Dyke,<sup>6</sup> is only visible in 78 per cent of normal encephalograms, angiography may have to be used in sellar

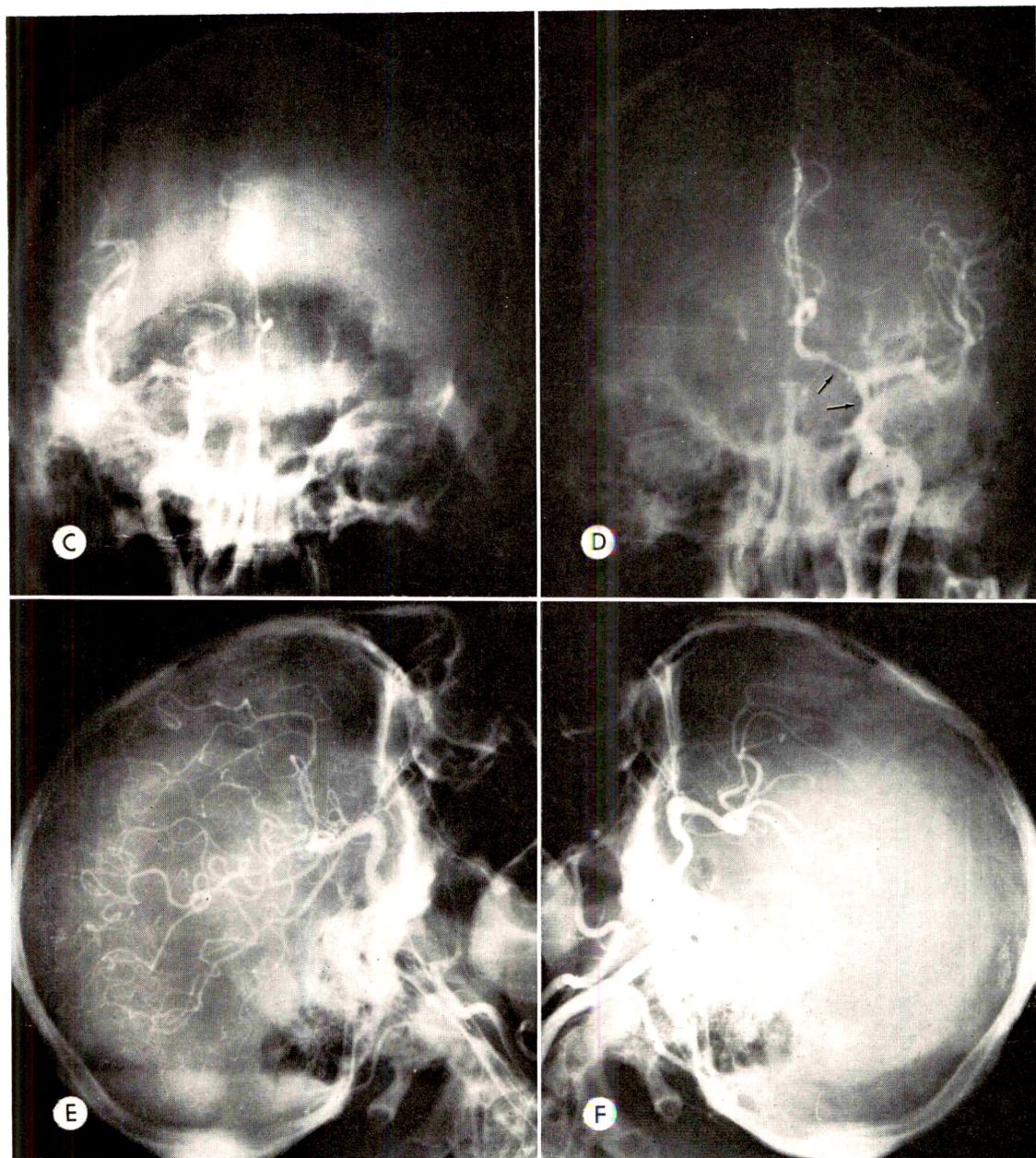


FIG. 9. (C and D) A comparison of the right and left anteroposterior angiogram in 1959 revealed a greater extent of the mass to the left, manifested by elevation of the left anterior cerebral artery (upper arrow), lateral bowing and thinning of the supraclinoid segment (lower arrow) and lateral displacement of the posterior intracavernous segment of the left carotid artery. (E and F) A comparison of the right and left lateral angiograms shows a slightly greater degree of opening of the left carotid siphon.



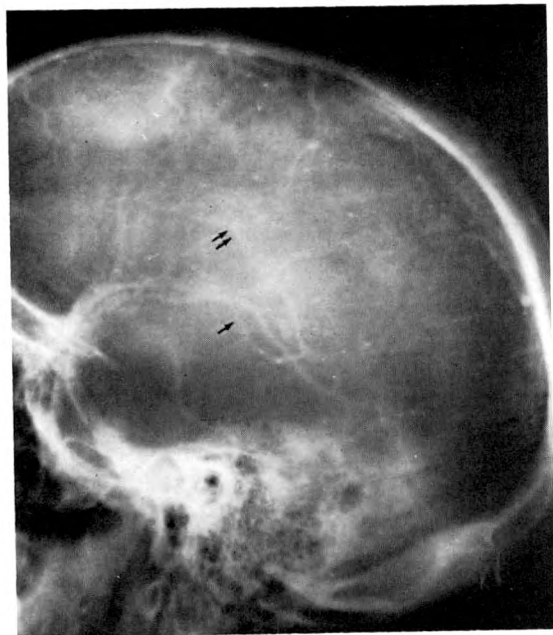


FIG. 10. Case VIII. Chromophobe, greatly cystic, pituitary adenoma in a 35 year old male. Left lateral angiogram demonstrates the marked upward and backward deflection of the basilar vein (arrow), intracerebral vein and venous angle (double arrow).

tumor suspects if the cistern is not demonstrated or if there is no evidence of indentation of the third ventricle.

The present policy of our neurosurgical department in the preferred use of pneumography and/or angiography in cases of pituitary adenomas is not a fixed one. If there is evidence of a hypersecreting eosinophilic adenoma, and if the clinical or pneumographic findings do not indicate a marked suprasellar extension, arteriography is omitted. If there is suspicion of a very large chromophobe adenoma on the bases of roentgenologic or clinical findings, arteriography will be carried out first or ventriculography may occasionally be decided upon. However, in the majority of cases pneumography will precede angiography. Should fast-progressing symptoms indicate a possible pituitary hemorrhage or if an aneurysm simulating a chromophobe pituitary adenoma, particularly in the presence of a subarachnoid hemorrhage, is suspected, angiography is the

method of first choice. In this connection it should be mentioned that a chromophobe pituitary adenoma or hemorrhage into a pituitary adenoma may cause subarachnoid hemorrhage and thereby simulate an aneurysm.<sup>2,18</sup>

Since 1951, 22 patients with suspected pituitary adenomas were admitted to the Neurosurgical Section for surgical intervention. Twenty-one patients showed signs of suprasellar extension, with spontaneous recovery in 1 case of hemorrhage into a presumably chromophobe adenoma.<sup>18</sup> In 18 cases, a chromophobe adenoma was histologically verified, although 3 patients showed signs of acromegaly. In 1 patient, an eosinophilic adenoma, in another a chordoma, and in a third a craniopharyngioma were the causes of the chiasmal syndrome. We employed pneumography routinely in all cases admitted for neurosurgical intervention since 1952, although additional angiography was occasionally used. A policy of combining both methods in most cases of suprasellar extension was adopted in more recent years. In our experience with pituitary adenomas and extrasellar extension of moderate to marked degree, both methods showed positive and complementary valuable findings. Since our material does not lend itself to a comparative statistical analysis, we merely selected 8 representative cases to demonstrate the various pneumographic and angiographic manifestations (Fig. 3 through 10).

#### SUMMARY

1. The roentgenological and surgical treatment of pituitary adenomas and of lesions which may simulate them clinically as well as on plain skull roentgenograms such as craniopharyngiomas, meningiomas, chordomas, carcinomas and aneurysms with extension into sellar and parasellar space, requires an early and exact determination of the size of the lesion, the predominant direction of its extension and, if possible, of its etiology.

2. In most instances, the roentgenograms and clinical findings do not give a definite

due as to the etiology of a tumor occupying the sella turcica or any exact information as to the size and direction of its extrasellar extension.

3. Cerebral pneumography and angiography are the only methods which permit an accurate estimate of intracranial tumor extension of pituitary adenomas, and should be performed in all pituitary tumor suspects before radiation therapy or surgical intervention is contemplated.

4. The neuroradiological diagnosis of expanding pituitary neoplasms requires not only familiarity with the normal and pathological topographical as well as roentgenological anatomy of the sella turcica and its adjacent structures, but also knowledge of the differential diagnostic characteristics of other lesions in the sellar area and also some awareness of the clinicopathological manifestations of pituitary tumor expansion. Although all these elements could hardly be discussed completely or in detail, some aspects necessary for a fuller understanding of the various phenomena of extrasellar extension of pituitary adenomas and the problems of their differential diagnosis were considered.

5. Based on available data of the literature and our own experience, the elements of the pneumographic and angiographic diagnosis of pituitary adenomas are illustrated and described, particularly in respect to the various pathways of suprasellar extension. A discussion of the various, partially controversial opinions expressed in the literature concerning the relative values of either method is added.

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## ARTERIOVENOUS SHUNTING IN CEREBROVASCULAR OCCLUSIVE DISEASE

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THE presence of early venous filling of intracerebral vessels usually indicates primary or secondary neoplasm of the brain or arteriovenous malformation. Other less common causes are acute inflammatory diseases of the brain<sup>7</sup> and well encapsulated intracerebral abscess.<sup>4</sup> On rare occasion, a superficial draining cortical vein may fill relatively early in the absence of any pathologic process. When the order of filling is sequential, however, this is of no significance; that is, the frontal veins fill prior to the parietal veins.<sup>5</sup>

Recently, we have seen 4 cases of cerebrovascular occlusive disease with early venous filling. We are able to find few previous reports in the literature describing this phenomenon.<sup>2,7</sup> Because of the implications of recognizing this important finding, and differentiating it from other more common causes of early venous filling, we feel that it is worthwhile to report our findings in several cases.

### REPORT OF CASES

**CASE 1. Middle Cerebral Artery Occlusion.** C.H., a 65 year old Negro male was admitted to the Boston City Hospital unable to move his right side. Twenty years ago, the patient was discharged from the Army, and placed on a pension for "dizzy spells." One year ago, he had been struck by an automobile and rendered unconscious. On awakening, right leg weakness was noted, but was minimal in degree. This did not progress or regress, and the patient was able to walk with no difficulty. Three weeks prior to admission, he suffered an episode of right sided hemiparesis which cleared slowly at home on no specific therapy.

On admission, he showed a right homonymous hemianopsia and right supranuclear facial

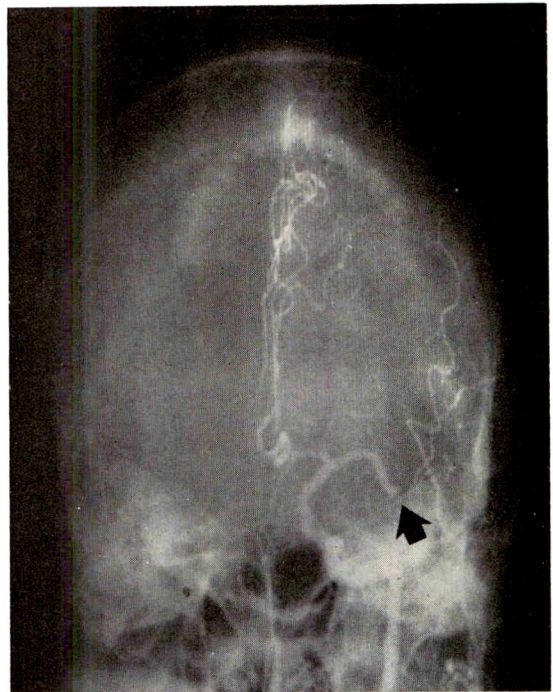


FIG. 1. Case 1. Left middle cerebral artery occlusion. Left anteroposterior common carotid angiogram shows occlusion of the M1 segment of the middle cerebral artery (arrow). The anterior cerebral artery is in the midline.

weakness. His eyes were deviated to the left. There was right sided hyperreflexia and a right extensor plantar response.

Spinal fluid was clear with an opening pressure of 150 mm. of water. Hematologic and chemistry studies were normal.

**Left Carotid Angiography.** Left common carotid angiography was performed. The anteroposterior view (Fig. 1), arteriographic phase, showed complete occlusion of the middle cerebral artery at its M1 segment. An abortive attempt at collateral circulation from the anterior cerebral artery was seen. There was no definite shift of midline structures. The lateral

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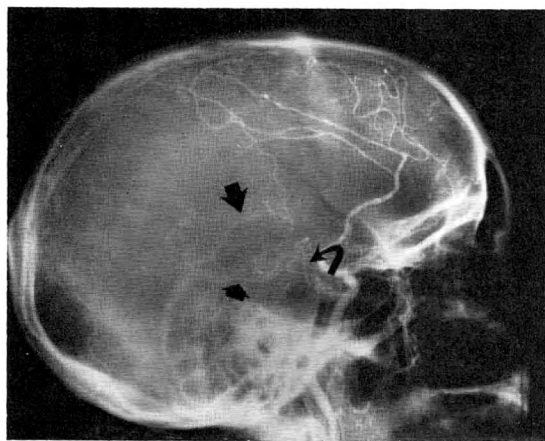


FIG. 2. Case I. Left middle cerebral artery occlusion. Left lateral carotid angiogram. The arteriographic phase shows complete occlusion of the middle cerebral artery with hypertrophied lenticulostriate and anterior choroidal vessels (curved arrow). The lower arrow points to the basal vein of Rosenthal; upper arrow denotes the internal cerebral vein. This illustrates simultaneous filling of both arteries and veins (1 second film).

view, arteriographic phase, at 1 second (Fig. 2) showed no filling of the middle cerebral artery group of vessels. The lenticulostriate arteries were hypertrophied, as well as the anterior choroidal artery, with a resultant "blush-like" pattern. The basal vein of Rosenthal and the internal cerebral vein were filled.

A lateral angiogram taken at 4 seconds (Fig. 3), showed retrograde filling from the basal vein of Rosenthal and internal cerebral vein to

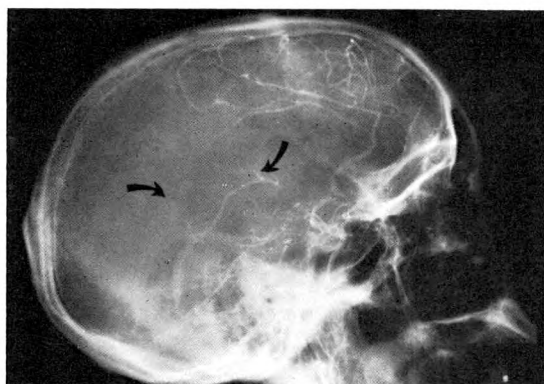


FIG. 3. Case I. Left middle cerebral artery occlusion. Left lateral common carotid angiogram. The arteriographic phase at 2 seconds shows retrograde filling of the septal and striothalamic veins (upper arrow), as well as reversal of flow in the inferior longitudinal sinus (lower posterior arrow).

the septal and striothalamic veins as well as reverse filling of the inferior longitudinal sinus.

*Comment.* The obvious presence of filling of the basal vein of Rosenthal and the internal cerebral vein at 1 second, simultaneously with the arteriographic phase was well demonstrated here. Serial angiograms furthermore showed retrograde venous flow in the internal cerebral vein to fill the septal and striothalamic veins. The reversal of flow was also present in the inferior longitudinal sinus. The arterial "blush" was clearly due to hypertrophied lenticulostriate vessels as well as the anterior choroidal artery.

*CASE II. Incomplete Occlusion of the Pericallosal Artery.* B.G., a 64 year old white diabetic hypertensive female, entered the Boston City Hospital, complaining of periods of memory loss.

On physical examination, her orientation and recall were appropriate for her age. The cranial nerves were unremarkable. The left plantar reflex was consistently flexor, the right variable. There was some right sided weakness.

Laboratory examinations were unremarkable except for persistently elevated blood sugar. Lumbar puncture showed an opening pressure of 195 mm. of water, with a protein of 73 mg. per cent.

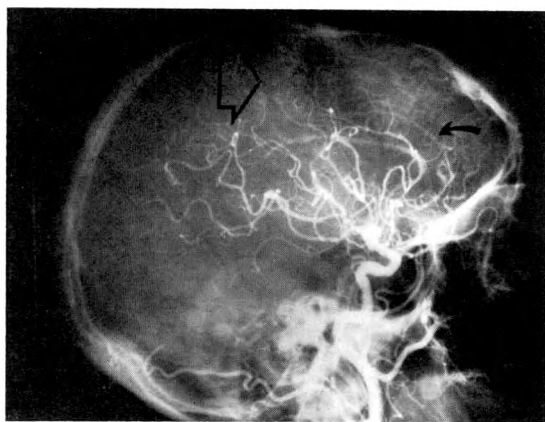


FIG. 4. Case II. Incomplete occlusion of the left pericallosal artery. Left lateral common carotid angiogram. The arteriographic phase shows scalloping of the pericallosal artery. The callosal marginal branch (small arrow) is hypertrophied. There is a vascular "blush" (large arrow) in the parietal region with early venous filling.

*Left Common Carotid Angiography.* Left common carotid angiography was performed. The lateral arteriographic phase (Fig. 4) showed marked narrowing and scalloping of the pericallosal artery. The callosal marginal branch was hypertrophied and showed a fine arterial blush of collateral vasculature somewhat well defined. There was simultaneous filling of draining frontoparietal veins with filling of the sagittal sinus. The middle cerebral artery also showed some irregularity of its lumen, consistent with atherosclerosis. A close-up study of the collateral vessels (Fig. 5), showed the draining cortical veins and sagittal sinus filling.

The anteroposterior angiogram demonstrated no shift of mid-line structures.

The angiographic findings, while suggestive of only vascular occlusive disease, could not exclude an infiltrating glioma on the basis of early venous filling.

A pneumoencephalogram failed to show any mass, but did show dilated sulci in the region of hypervascularity on angiography.

*Surgery and Hospital Course.* At exploratory surgery, no tumor was found. Biopsies in the area in question showed only gliosis. The patient's weakness improved somewhat, and she was discharged home with the diagnosis of cerebrovascular occlusive disease.

*Comment.* This striking example of partial occlusion of the pericallosal artery with collateral circulation was confused with a

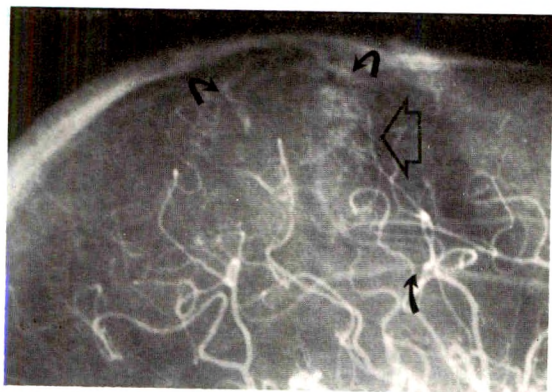


FIG. 5. Case II. Incomplete occlusion of the left pericallosal artery. Close-up view of Figure 4. Lower arrow points to an area of marked narrowing of the pericallosal artery. The vascular collateral "blush" is seen to good advantage here (large arrow). There is simultaneous fill of the draining parietal veins (curved arrows) into the sagittal sinus.



FIG. 6. Case III. Right middle cerebral artery occlusion. Right anteroposterior common carotid angiogram showing complete occlusion of the M1 segment of the middle cerebral artery (arrow).

glioma on the basis of early venous filling. The vascular blush of the collateral channels was composed of even calibered vessels. The venous drainage was at the periphery rather than within the confines of the network of anastomosing arteries. There was no evidence of a mass lesion as evidenced by the absence of displacement of mid-line structures on angiography and the supporting air study.

*CASE III. Middle Cerebral Artery Occlusion.* M.M., a 52 year old white female, entered the Boston City Hospital with the history of progressive weakness of her left side of a few months' duration.

Six months prior to admission, she had sustained head injuries in an automobile accident, but was not hospitalized.

On physical examination, she was noted to have a left sided hemiparesis, more marked in the arm with marked aphasia and a supranuclear facial palsy. Spinal puncture was normal.

*Right Common Carotid Angiography.* The anteroposterior arteriographic phase (Fig. 6) showed complete occlusion of the M1 segment





FIG. 7. Case III. Right middle cerebral artery occlusion. Right lateral common carotid angiogram. The injection is still in the arteriographic phase, yet the basal vein of Rosenthal (lower arrow) and the internal cerebral vein (upper arrow) are already filled. There is a slight "blush" in the middle cerebral artery distribution secondary to hypertrophied lenticulostriate vessels.

of the middle cerebral artery. The anterior cerebral artery was not shifted. The lateral arteriographic phase (Fig. 7) at 4 seconds from the beginning of injection demonstrated early filling of the basal vein of Rosenthal and the internal cerebral vein. There was a slight "blush" of hypertrophied lenticulostriate and anterior choroidal vessels. A close-up view (Fig. 8) demonstrated the early venous filling to better advantage.

*Comment.* This case of occlusion of the middle cerebral artery, as Case 1, vividly

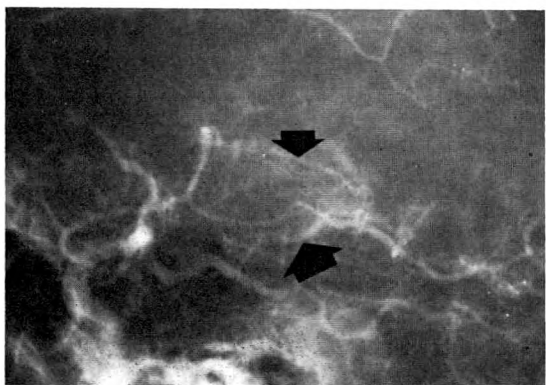


FIG. 8. Case III. Right middle cerebral artery occlusion. Close-up view of Figure 7 illustrates the early venous filling of the basal vein of Rosenthal (lower arrow) and the internal cerebral vein (upper arrow).

demonstrated the early venous filling of the basal vein of Rosenthal and the internal cerebral vein. Retrograde flow in the internal cerebral vein and inferior longitudinal sinus could not be seen, however.

*CASE IV. Middle Cerebral Artery Occlusion.* R.B., a 54 year old Negro female, was admitted to the Boston City Hospital complaining of frontal headaches, lethargy, and forgetfulness, of one year's duration.

On physical examination, it was noted that the patient was not fully oriented. There was a left supranuclear facial palsy. Deep tendon reflexes were slightly more active on the left. Lumbar puncture showed a protein of 90 mg. per cent.

*Right Carotid Angiography* (Fig. 9). There was a large vascular tumor occupying most of the anterior temporal lobe. The circulation was quite slow, and no demonstrable early venous filling was seen. The vascular pattern was interpreted as being consistent with a glioma.

*Surgery and Hospital Course.* At surgery, a large deep mid-temporal vascular astrocytoma, Grade 2, was excised through an incision in the superior temporal gyrus. Several vessels were clipped during the procedure.

Because of a downhill course postoperatively, a second carotid angiography was performed on the right side on the third postoperative day.

*Second Right Common Carotid Angiography* (Fig. 10). The lateral angiogram showed occlu-

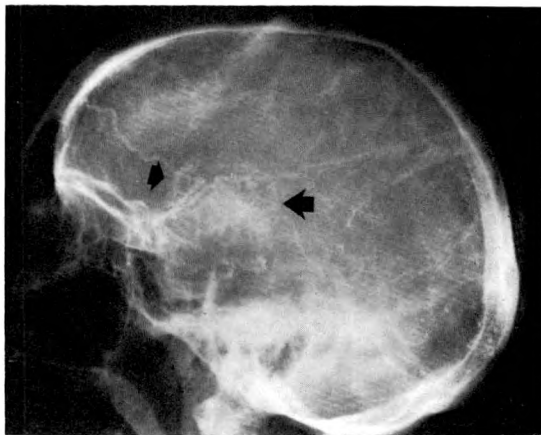


FIG. 9. Case IV. Right middle cerebral artery occlusion. Preoperative study. Right lateral common carotid angiogram. The venous phase shows a vascular tumor (arrows) arising in the temporal lobe. The circulation in the tumor was slow.

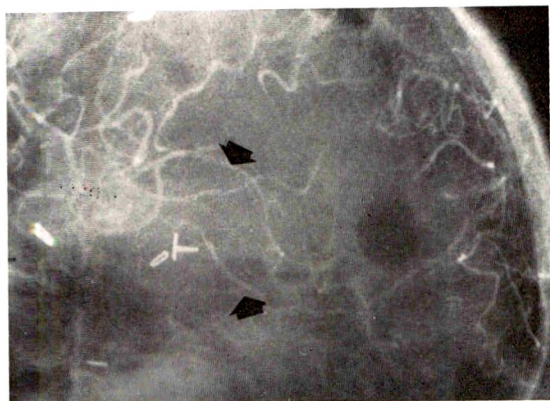


FIG. 10. Case IV. Right middle cerebral artery occlusion. Postoperative study. Right lateral common carotid angiogram. The arteriographic phase shows also simultaneous filling of the basal vein of Rosenthal (lower arrow) and the internal cerebral vein (upper arrow).

sion of the middle cerebral artery (anteroposterior angiogram verified that a clip was occluding the M1 segment). The 1 second study in the arteriographic phase showed simultaneous visualization of the basal vein of Rosenthal and the internal cerebral vein. There was very little evidence of collateral circulation.

*Comment.* While the presence of neoplasm (presumably completely excised) complicates this case, it is noteworthy that the angiogram made preoperatively showed no early venous filling. One can assume, similar to Cases I and III, that the middle cerebral artery occlusion was the etiologic agent of the arteriovenous shunting here rather than neoplastic involvement.

#### DISCUSSION

The presence of early venous filling on cerebral angiography in gliomas and arteriovenous malformations has been reported by Goree and Dukes.<sup>1</sup> They stress the orderly appearance of the arterial feeding supply in malformations as opposed to the tortuous arterial supply to a glioma. Furthermore, avascular spaces are not seen in malformations. Metastatic disease to the brain may, on occasion, show a hypervascular pattern on angiograms and simulate glioma. Meningiomas have been said not to show early arteriovenous shunting. There

is a group of vascular meningiomas, however, which may show early venous filling with a vascular pattern simulating gliomatous vasculature.<sup>6</sup>

Less well known causes of early venous filling are acute inflammatory cerebral processes,<sup>7</sup> well encapsulated abscesses,<sup>4</sup> and vascular occlusions.<sup>2,7</sup>

The mechanism in the 4 cases described above is not clear. It does appear, however, that the collateral arterial routes fill preferentially earlier than comparable vessels in the normal cerebral vasculature. The demand of blood in the ischemic segment of the brain appears to siphon off blood from the proximal unoccluded vessels at a proportionally faster rate. The venous drainage, then, although filling earlier than in other unaffected areas in the hemisphere, is supplied with blood (and thus with the injected opaque material) at an earlier time. The draining veins appear to be normal in contour. The early venous filling, it would appear, is a relative phenomenon, depending on the degree of preferential filling rate of the collateral arterial routes.

Differentiation of this early venous filling from other shunting processes may be difficult. The presence of occlusive disease, either complete or partial, should arouse one's suspicion as to the etiology. There may or may not be an arterial blush representing collateral circulation. The feeding vessel may be large. The presence of a marked blush of collateral routes may cause considerable diagnostic difficulty with neoplasms, as pointed out by Leeds and Abbott.<sup>3</sup> The caliber of these vessels is small, multiple, and orderly. The number of draining venous channels in our cases of occlusive disease was smaller than the number of arterial collateral routes. Furthermore, the venous channels tended to be localized outside of the confines of the hypervascular arterial collateral routes. The arterial-venous connections could not actually be seen. In 2 of the 3 cases of middle cerebral artery occlusion, the venous drainage was so rapid that there was de-



monstrable retrograde venous filling instead of the normal antegrade flow. All 3 showed no mass effect.

The following points, then, would seem to categorize the early venous filling in cerebral occlusive disease:

1. Angiographic evidence of occlusive disease, either partial or complete.
2. The arterial blush of the collateral circulation, if present, is small, multiple, and orderly.
3. The number of venous channels is smaller than the number of arterial channels. The venous drainage tends to be outside of the hypervascularized arterial collateral routes.
4. There may be retrograde venous filling.
5. Lack of a localized mass, although brain edema may cause a mid-line shift.

While the lack of experience with early venous filling in occlusive disease of the cerebral hemisphere makes it difficult to establish absolute criteria of differential diagnosis, we do believe that the above 5 points are of considerable value in distinguishing early venous filling in cerebrovascular occlusive disease from other causes of venous shunting in the brain. The addition of a gas study in situations such as Case II will, of course, be of value in confirming the absence of a mass lesion.

#### SUMMARY

Four cases of early venous filling in cere-

brovascular occlusive disease are reported.

Some criteria for differential diagnosis from neoplasm and arteriovenous malformations are presented.

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## CRANIAL COLLATERAL PATHWAYS IN STROKE SYNDROME

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CEREBRAL angiography has been extensively used in the study of stroke syndrome, particularly since the emergence of corrective vascular surgery. This presentation is limited to the cranial collateral pathways, as the numerous collateral pathways of brachiocephalic occlusive disease have been well discussed in recent publications.<sup>8,10,11,16,18</sup> The illustrative cases cited below have been selected from two large charity institutions and one large private teaching hospital, with a combined total of more than 500 angiographies per year on patients with a history of stroke or transient ischemic attack.

The cranial collateral pathways include:<sup>11,16</sup> (1) the circle of Willis; (2) end artery anastomoses over the surface of the brain from the anterior, middle and posterior cerebral arteries; and (3) the external to internal carotid artery with combinations frequently observed. Bilateral occlusive or stenotic disease is common. Serial filming over a period of at least 6 seconds has been routine in each of these institutions, as the pathways are frequently not demonstrable on the earliest films, nor would they be apparent on single film technique. In the event of a stroke, the collateral pathways are immediately available, although flow through them will increase with demand (Case VIII, Fig. 8, A and B).

The occurrence of collateral blood flow in the brain is dependent on the head of pressure in the vessels which supply an area. If the pressure is lowered by local occlusion or narrowing, then reversal of blood flow will occur.



FIG. 1. Case I. Right common carotid angiogram shows filling of the left anterior and middle cerebral arteries via the anterior communicating artery. The left internal carotid artery was occluded at its origin in the neck.

The causes of this are listed as follows:<sup>15</sup>

- A. Narrowing of lumen
  1. Atherosclerosis (plaque or thrombus)
  2. Embolus
  3. Congenital atresia or stenosis
  4. Surgical ligation
  5. Compression of artery extramurally
    - (a) mass in neck or mediastinum
    - (b) arthritic spur
    - (c) digital compression

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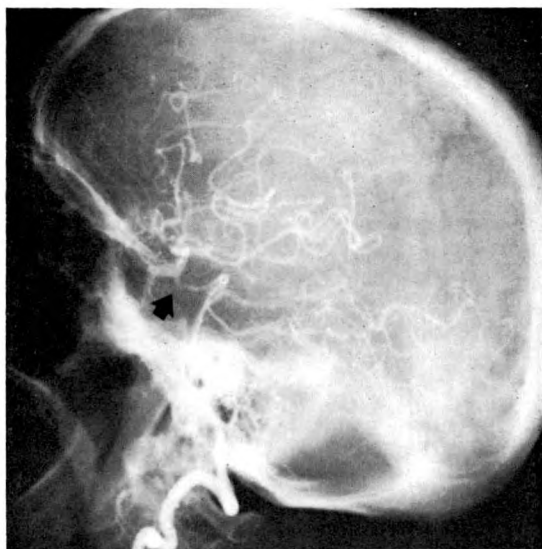


FIG. 2. Case II. Retrograde right brachial angiogram shows occlusion of the carotid artery on the right and the vertebral artery fills the basilar system. The anterior and middle cerebral arteries are filled from the basilar system via a large posterior communicating artery.



FIG. 3. Case III. Retrograde right brachial angiogram shows a hypoplastic right vertebral artery and collateral flow via the posterior communicating artery to fill both posterior cerebral arteries and retrograde fill of the basilar and both superior cerebellar arteries. There was atherosclerotic occlusion of the basilar artery immediately below the superior cerebellar arteries.

#### B. Dilatation of vessel lumen

1. Arteriosclerosis
2. Localized vasodilator effects

#### C. Vascular shunts

1. Carotid—cavernous fistula
2. Arteriovenous malformation

#### I. THE CIRCLE OF WILLIS

Thomas Willis<sup>19</sup> described the circle of Willis in 1684, and demonstrated its importance in the collateral circulation of the brain. Riggs and Griffiths<sup>14</sup> reported on the anatomic dissection of 1,647 brains in 1938 and subdivided their findings into 8 groups of variations of the circle of Willis. Alpers *et al.*<sup>1</sup> reported a study of 350 normal brains in which the circle of Willis was normal in only 52.3 per cent of the cases. The most frequent anomaly was a filiform caliber of one of the component vessels in 27.4 per cent of the circles. This was most often present in one or both posterior communicating arteries. The embryonic origin of the posterior cerebral artery from the in-

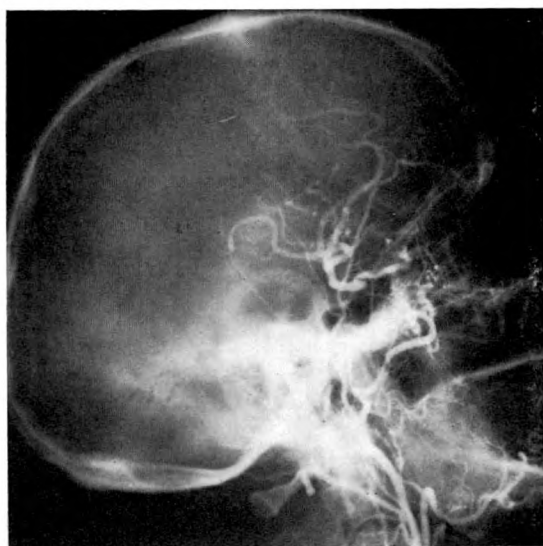


FIG. 4. Case IV. Left carotid angiogram shows occlusion of the internal carotid artery at its origin. Collateral flow via the ophthalmic artery from the internal maxillary artery fills the siphon and both the anterior and middle cerebral arteries. The right internal carotid artery was also occluded—a likely explanation of why the ophthalmic artery was the principal collateral and not overshadowed by the anterior communicating artery collateral.

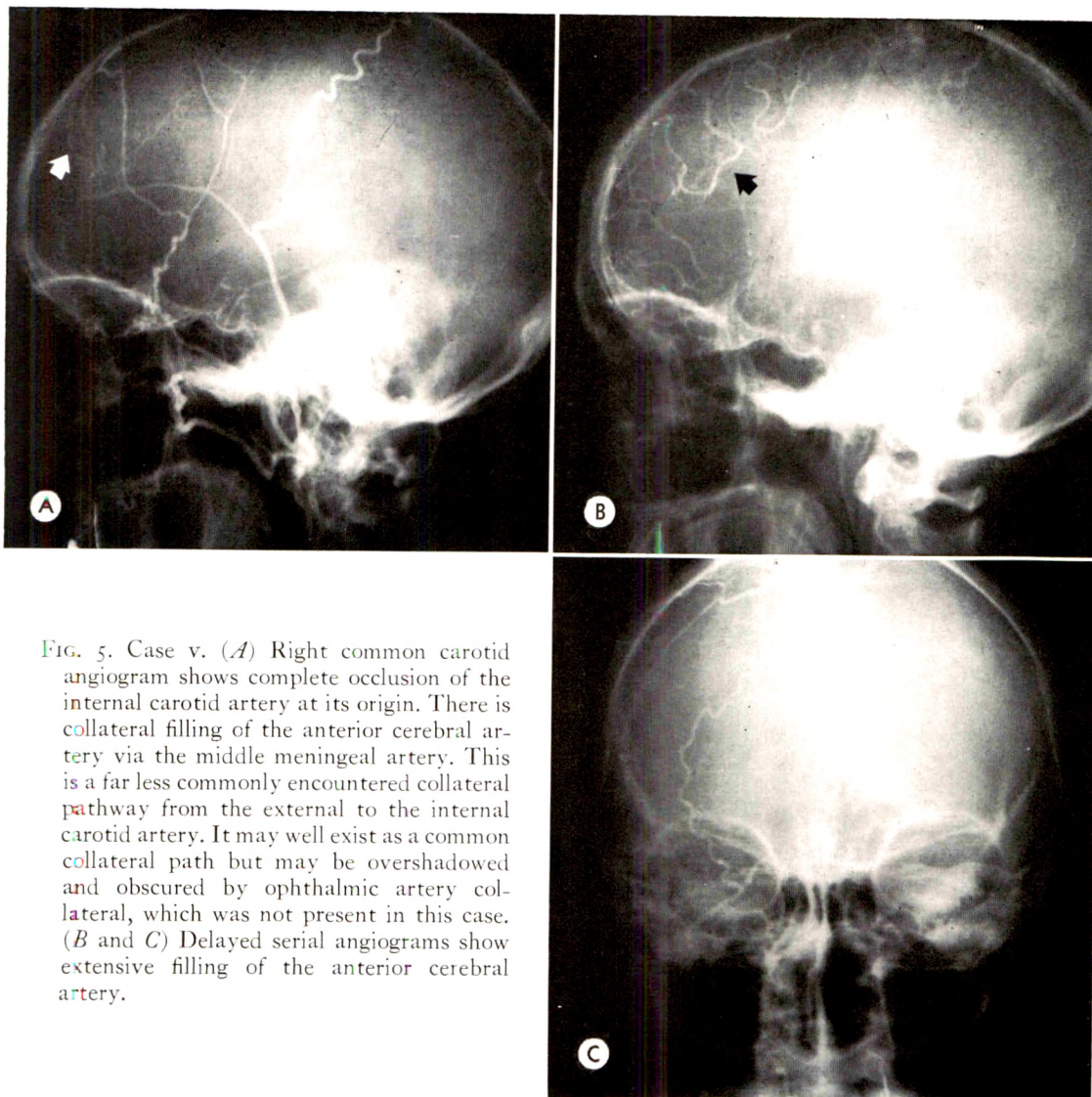


FIG. 5. Case v. (A) Right common carotid angiogram shows complete occlusion of the internal carotid artery at its origin. There is collateral filling of the anterior cerebral artery via the middle meningeal artery. This is a far less commonly encountered collateral pathway from the external to the internal carotid artery. It may well exist as a common collateral path but may be overshadowed and obscured by ophthalmic artery collateral, which was not present in this case. (B and C) Delayed serial angiograms show extensive filling of the anterior cerebral artery.

ternal carotid persisted in 14.6 per cent of the cases (Case I, Fig. 1; Case II, Fig. 2; and Case III, Fig. 3).

## II. EXTERNAL TO INTERNAL CAROTID ARTERY ANASTOMOSES<sup>2,4,10,11</sup>

This collateral flow is most commonly observed via retrograde flow through the ophthalmic branch of the internal carotid artery.<sup>3-6,10,13</sup> The principal collateral branches of the external carotid artery include the internal maxillary, external maxillary, and the superficial temporal arteries in order of decreasing contribution. The

ophthalmic artery does increase in size in response to the additional flow<sup>11</sup> (Case IV, Fig. 4; and Case v, Fig. 5, A, B and C).

## III. ANASTOMOSES OVER THE SURFACE OF THE BRAIN<sup>4,7,12,17,18</sup>

These anastomoses over the surface of the brain, so-called meningeal or pial anastomoses between and among the major cerebral arteries, are frequently observed employing long serial technique.

1. *Anterior to middle cerebral artery collateral flow via pial anastomoses.* These pathways are usually noted extending from the



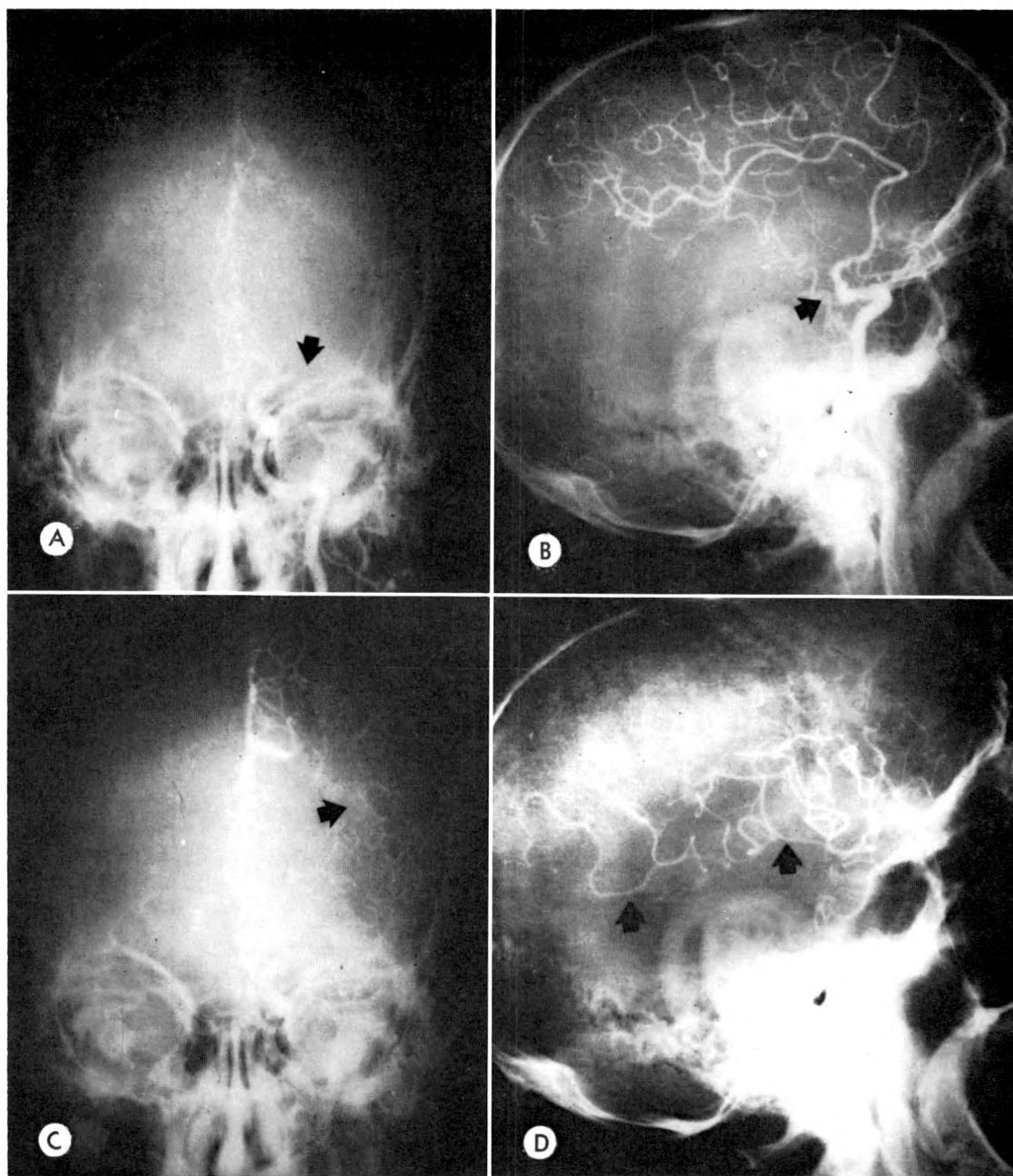


FIG. 6. Case VI. (A and B) Left common carotid angiograms show complete occlusion of the middle cerebral artery immediately distal to its origin. (C) Later anteroposterior serial angiogram shows filling of the middle cerebral artery from the anterior cerebral artery via pial anastomoses. (D) Still later lateral serial angiogram shows extensive retrograde collateral filling of the middle cerebral artery back to its point of occlusion.

callosomarginal to the ascending frontoparietal and the sylvian group of vessels. This is a common pathway in occlusive disease, either segmental or involving the major portion of the vessel proximal to

the trifurcation (Case VI, Fig. 6, A-D; and Case VII, Fig. 7, A and B).

2. *Middle to anterior cerebral artery via pial anastomoses.* The vessels contributing to this are the ascending frontoparietal and

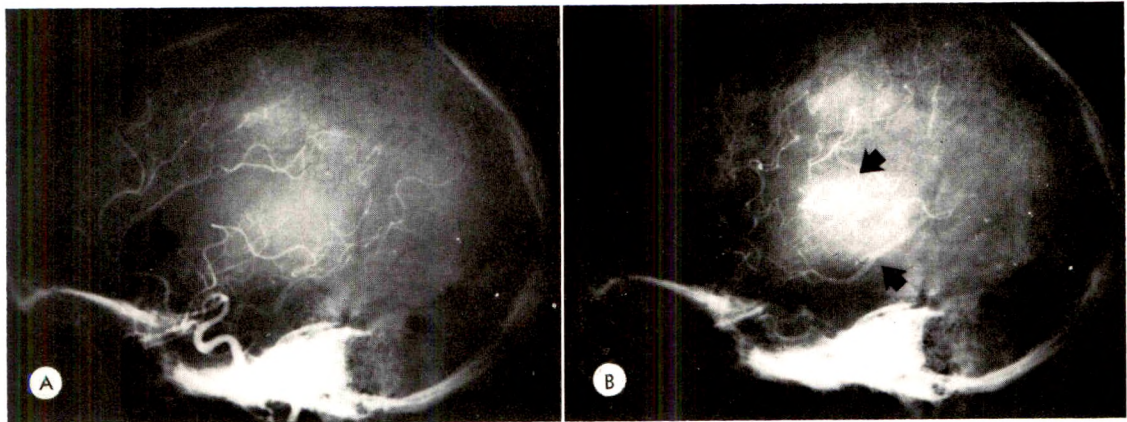


FIG. 7. Case VII. (A) Another patient examined by right common carotid angiography shows absence of vessels in the ascending frontoparietal and posterior parietal regions. (B) This segmental occlusion<sup>9</sup> of the ascending frontal branch of the middle cerebral artery is demonstrated by delayed retrograde collateral filling from the anterior cerebral artery via pial anastomoses. The basal vein of Rosenthal appears at the normal time in the study while retrograde arterial collateral flow is still evident.

anterior branches of the sylvian group which will ascend around the convexity of the brain and course supero-medially to the occluded portion of the anterior cerebral vessel. This is uncommon, as we have encountered occlusion of the anterior cerebral

on only rare occasions, and a more common pathway would be across the midline via the anterior communicating artery (Case VIII, Fig. 8, A and B; and Case IX, Fig. 9, A and B).

### 3. Posterior to anterior cerebral artery

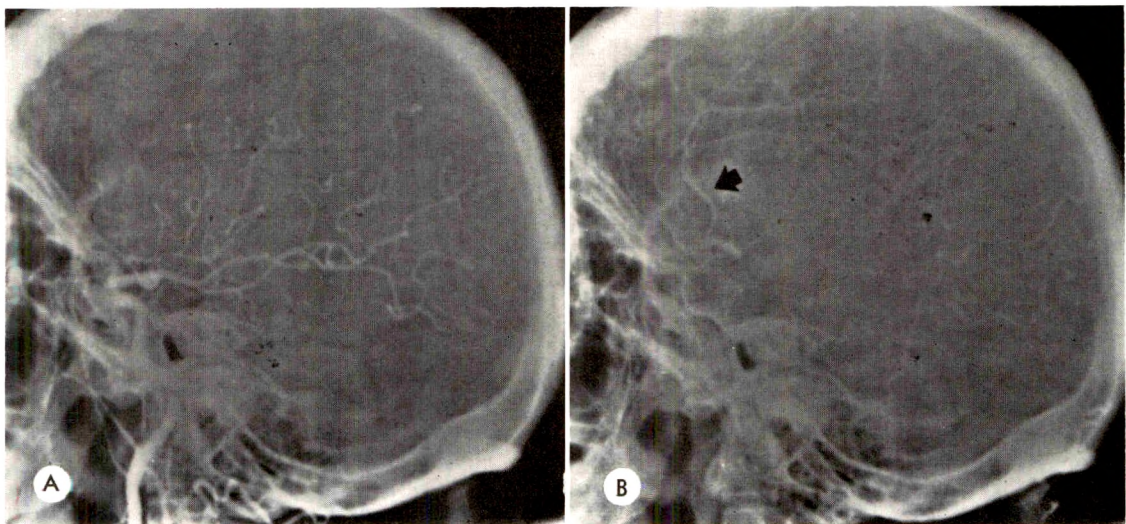


FIG. 8. Case VIII. This 68 year old white female with a negative previous history suffered a "stroke" while visiting in the hospital. Physical examination showed complete coma, areflexia and atrial fibrillation. Embolus was postulated. Left carotid angiography only 45 minutes after onset of stroke showed complete occlusion of the left internal carotid artery in the siphon (not shown). (A) Right carotid angiogram shows anterior cerebral artery occlusion. (B) Delayed angiogram shows retrograde collateral filling of the anterior from the middle cerebral artery via pial anastomoses. There was no opportunity for flow across the anterior communicating artery as the opposite left carotid artery was occluded in the siphon. The unusual opportunity for examination less than 1 hour following stroke demonstrates the immediate availability of the collateral channels.



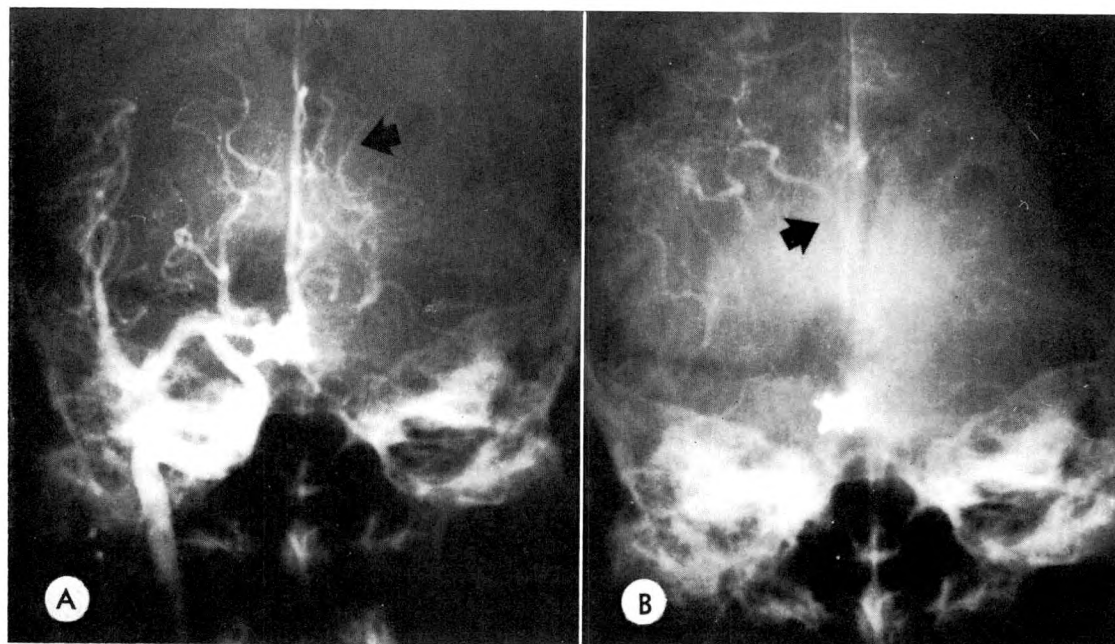


FIG. 9. Case IX. Right anterior cerebral artery clipped at surgery distal to anterior communicating artery. (A) Right common carotid angiogram shows filling of the right posterior and middle cerebral arteries and left anterior cerebral artery via the anterior communicating artery. (B) Delayed angiogram shows retrograde filling of the right anterior cerebral artery from the right middle cerebral artery via pial anastomoses, with retrograde filling to the point of surgical occlusion.

*collateral flow via pial anastomoses.* Anastomosis exists between anterior occipital artery and occipital branch of posterior cerebral artery (Case X, Fig. 10).

4. *Posterior to middle cerebral artery collateral flow via pial anastomoses.* An anas-

tomotic pathway exists via temporal branches of the middle cerebral artery and posterior cerebral artery (Case XI, Fig. 11, A, B and C).

5. *Anterior and middle cerebral arteries to posterior cerebral artery via pial anastomoses.* The same pathway as above (4) reverses its flow in occlusive disease of the posterior cerebral vessels (Case XII, Fig. 12, A, B and C).

#### IV. COMBINED COLLATERAL PATHWAYS

Combined pathways are far more common than isolated collateral pathways, and

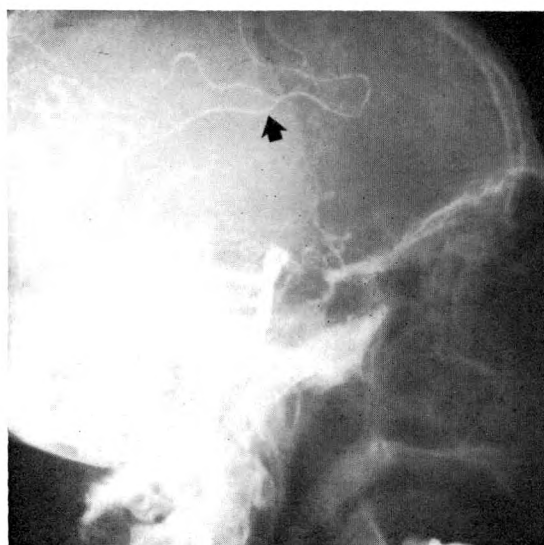


FIG. 10. Case X. Unintentional left vertebral injection (left internal carotid occlusion and right internal carotid stenosis later demonstrated) shows excellent filling of the basilar and posterior cerebral arteries and retrograde filling of the anterior cerebral artery.

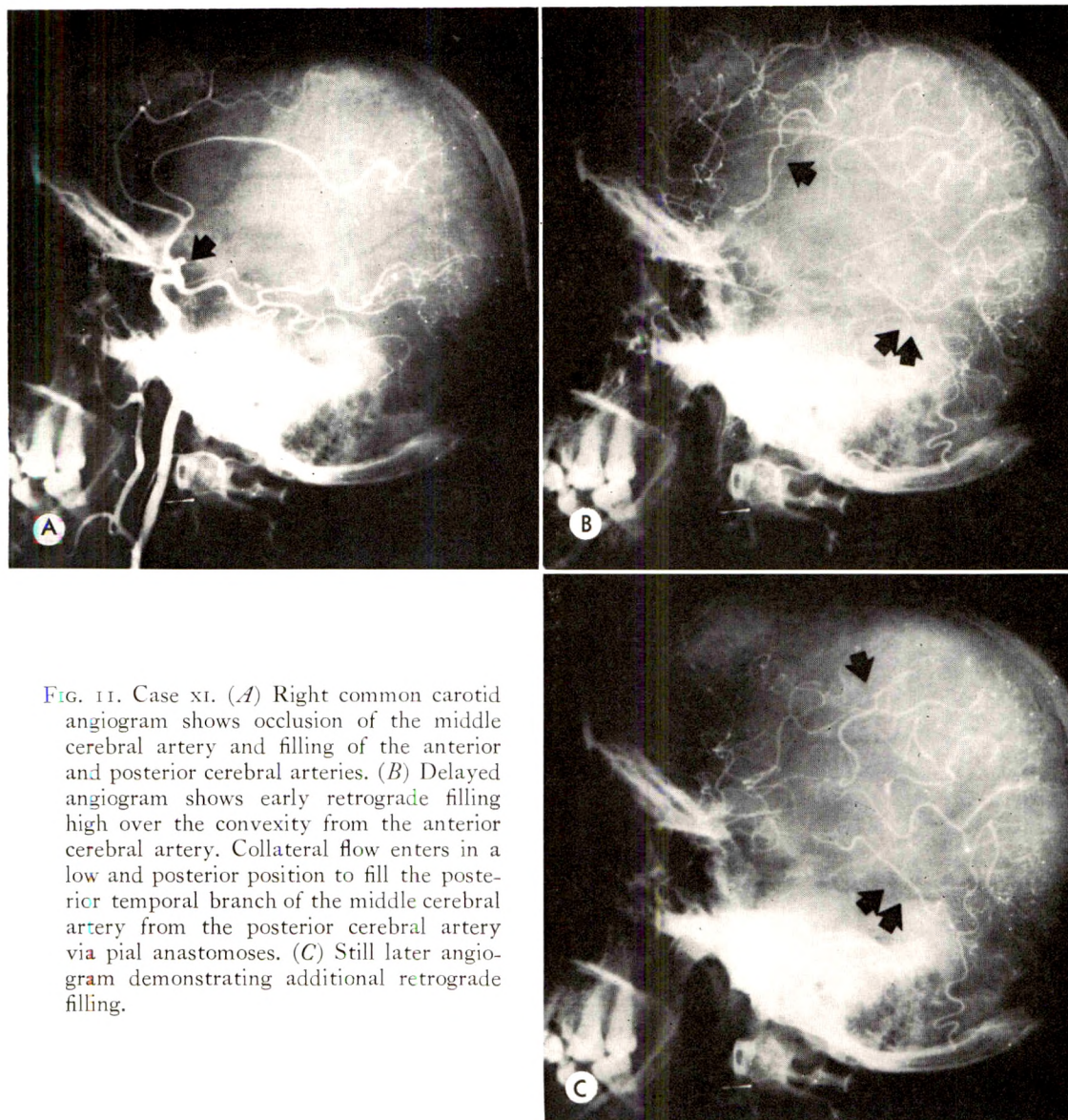


FIG. 11. Case XI. (A) Right common carotid angiogram shows occlusion of the middle cerebral artery and filling of the anterior and posterior cerebral arteries. (B) Delayed angiogram shows early retrograde filling high over the convexity from the anterior cerebral artery. Collateral flow enters in a low and posterior position to fill the posterior temporal branch of the middle cerebral artery from the posterior cerebral artery via pial anastomoses. (C) Still later angiogram demonstrating additional retrograde filling.

demonstration of the different collateral pathways is, of course, dependent upon the type of examination employed, direct carotid injection, direct vertebral injection, retrograde brachial injection, or aortic root catheter injections. Many, if not most, cases of unilateral internal carotid occlusion will have ipsilateral collateral flow via the ophthalmic artery and simultaneous contralateral collateral flow via the anterior communicating artery. These are more commonly observed than the posterior

communicating artery collateral flow for the following reasons: (a) more carotid injections are done than vertebral-basilar opacifications; (b) the known variability of the size and origin of the vertebral arteries; and (c) known high incidence of variability of the posterior communicating arteries (Case XIII, Fig. 13, A, B and C).

#### SUMMARY

1. Cranial collateral circulation is consistently demonstrable by serial filming.



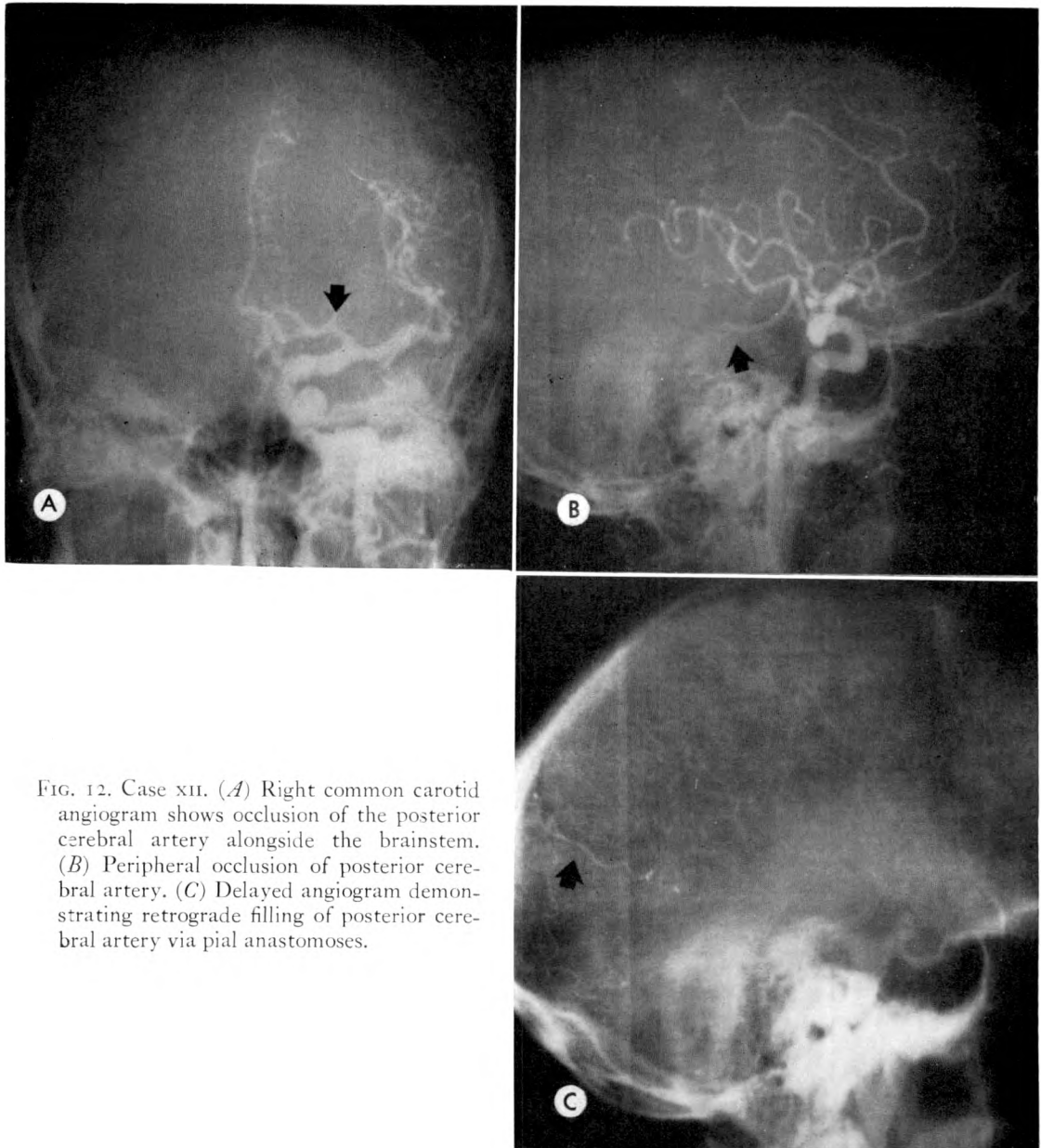


FIG. 12. Case XII. (A) Right common carotid angiogram shows occlusion of the posterior cerebral artery alongside the brainstem. (B) Peripheral occlusion of posterior cerebral artery. (C) Delayed angiogram demonstrating retrograde filling of posterior cerebral artery via pial anastomoses.

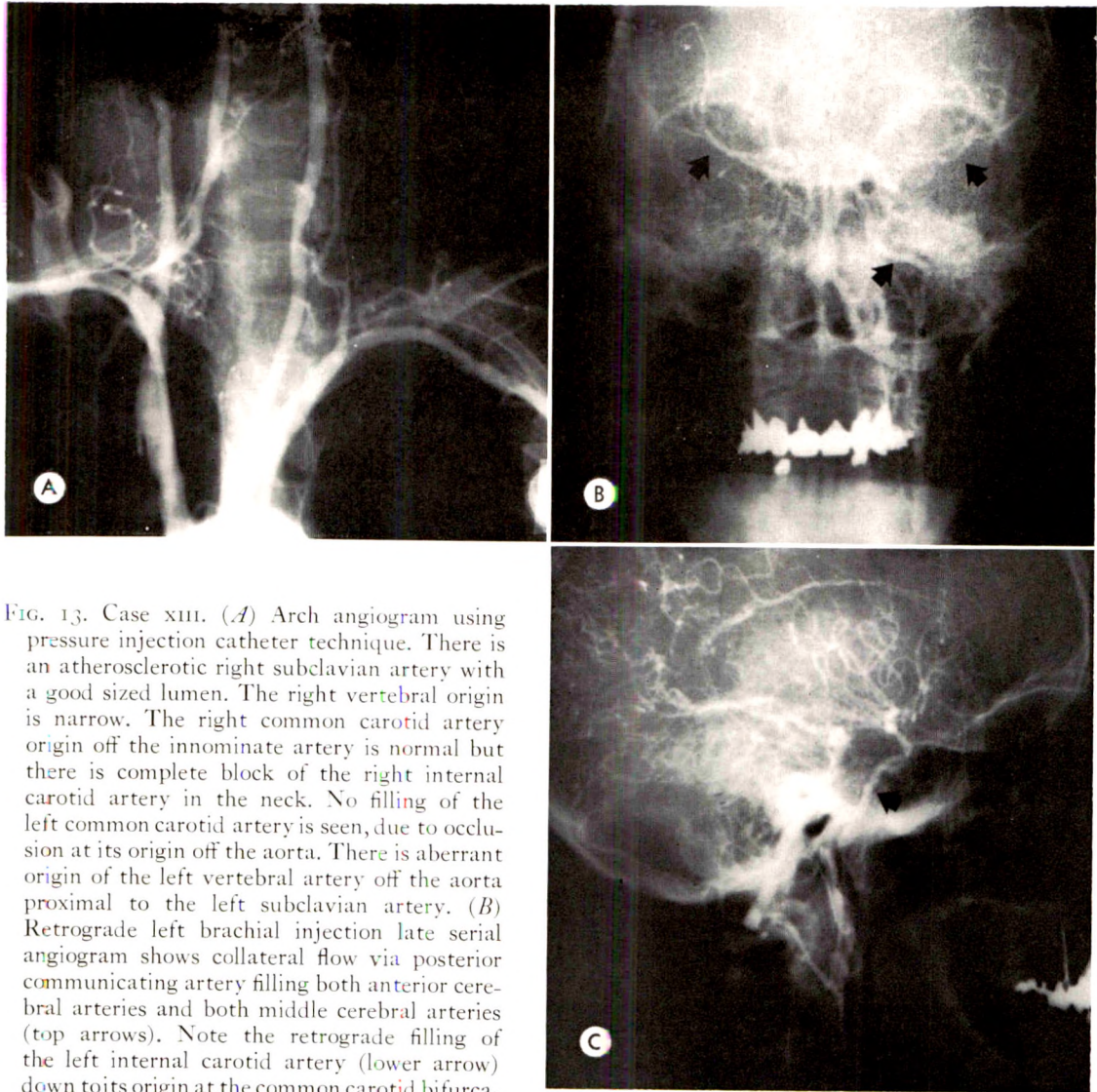


FIG. 13. Case XIII. (A) Arch angiogram using pressure injection catheter technique. There is an atherosclerotic right subclavian artery with a good sized lumen. The right vertebral origin is narrow. The right common carotid artery origin off the innominate artery is normal but there is complete block of the right internal carotid artery in the neck. No filling of the left common carotid artery is seen, due to occlusion at its origin off the aorta. There is aberrant origin of the left vertebral artery off the aorta proximal to the left subclavian artery. (B) Retrograde left brachial injection late serial angiogram shows collateral flow via posterior communicating artery filling both anterior cerebral arteries and both middle cerebral arteries (top arrows). Note the retrograde filling of the left internal carotid artery (lower arrow) down to its origin at the common carotid bifurcation and subsequent antegrade filling of the external carotid artery. (C) Lateral view of same (arrow on internal carotid artery). Note that there are also pial anastomoses with retrograde filling of the anterior cerebral artery from the posterior cerebral artery. This patient had a still later injection of his right common carotid artery (with known complete obstruction of the internal carotid artery) and the collateral flow occurred via the middle meningeal to the anterior cerebral artery as shown in Figure 5, A and B.



2. There are 3 principal cranial collateral pathways.

I. Circle of Willis.

II. External to internal carotid anastomoses via:

(a) Ophthalmic artery reversed flow (commonly observed)

(b) Middle meningeal branch of external carotid to the meningeal branches of the cerebral arteries (uncommonly demonstrated).

III. Over the surface of the brain, so-called meningeal or pial anastomoses between and among the 3 major cerebral arteries.

3. These collateral pathways exist awaiting demand, and enlarge as demand for flow rate and volume increases.

4. It is our impression that prognosis is related to the richness of demonstrable pathways.

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## ANEURYSM AS A CAUSE OF STROKE\*

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WHEN an intracranial aneurysm ruptures, there is usually intense spasm of the adjacent arteries. This local spasm tends to serve as a protective mechanism to reduce intracranial bleeding. At times, the spasm may be so intense as to cause nonfilling of one of the intracranial branches of the circle of Willis on angiography. If the occlusion is gradual enough, collateral circulation may develop to compensate for the reduced intracranial blood flow. The purpose of the authors is to show that: (1) arterial spasm secondary to a bleeding aneurysm may cause angiographic occlusion of an artery; (2) collateral blood flow may develop which will significantly influence the postoperative course of the patient; and (3) the radiologist must take special care to demonstrate the collateral pathways and alert the neurosurgeon to collateral vessels.

### HISTORY

The spasm that accompanies ruptured intracranial aneurysms has been shown to be most intense in the first 2 to 3 days following the bleeding.<sup>5</sup> The spasm may remain for variable periods of time but usually tends to subside after 3 to 4 weeks.<sup>4,6</sup> The grave prognostic significance of prolonged spasm has been pointed out by many investigators.<sup>11,13,14</sup> The influence of injected contrast material has been incriminated as an etiologic factor in spasm. However, the overwhelming weight of evidence shows that spasm is intimately associated with bleeding and traction or trauma to the arterial wall. Gradual relaxation of spasm can be demonstrated by serial angiography.<sup>6</sup> Logue,<sup>11</sup> in 1956, stated that the severe spasm may predispose to cerebral infarction and may make surgery for intra-

cranial aneurysms very dangerous. The postoperative morbidity of patients with spasm is increased.<sup>13</sup> Two patients who died in the postoperative period were reported by Fletcher *et al.*<sup>8</sup> as showing encephalomalacia in the area of distribution of the vessels in spasm without any evidence of significant residual hematoma, thrombosis, or intracerebral rupture. Pool *et al.*<sup>14</sup> have stated that the severe vasoconstriction may cause hemiplegia and serious cerebral edema that may prove fatal. They further stated that this spasm is reversible and may disappear on subsequent angiographic studies. The spasm may be so severe as to prevent an aneurysm from filling but no instances of vascular occlusion secondary to the spasm were recorded. Freidenfelt and Sundstrom<sup>9</sup> reported 5 cases of severe spasm. Rupture of intracranial aneurysms had occurred in 3 patients and they showed no evidence of occlusion. Two of the cases that were associated with head trauma showed vascular occlusion with collateral circulation flow patterns.

From the neurosurgical service of UCLA Center for the Health Sciences and Wadsworth V.A. Hospital, 3 patients were found with aneurysms arising from the first portion of the posterior cerebral artery. All were investigated by selective carotid angiography followed by surgery.

### REPORT OF CASES

CASE 1. A 23 year old left-handed male was admitted on January 25, 1966, with the chief complaint of severe headaches. In late December 1965, he developed intermittent headaches of sudden onset, beginning in the suboccipital region and becoming generalized, occurring almost daily, lasting 2 to 3 minutes, and stopping suddenly.

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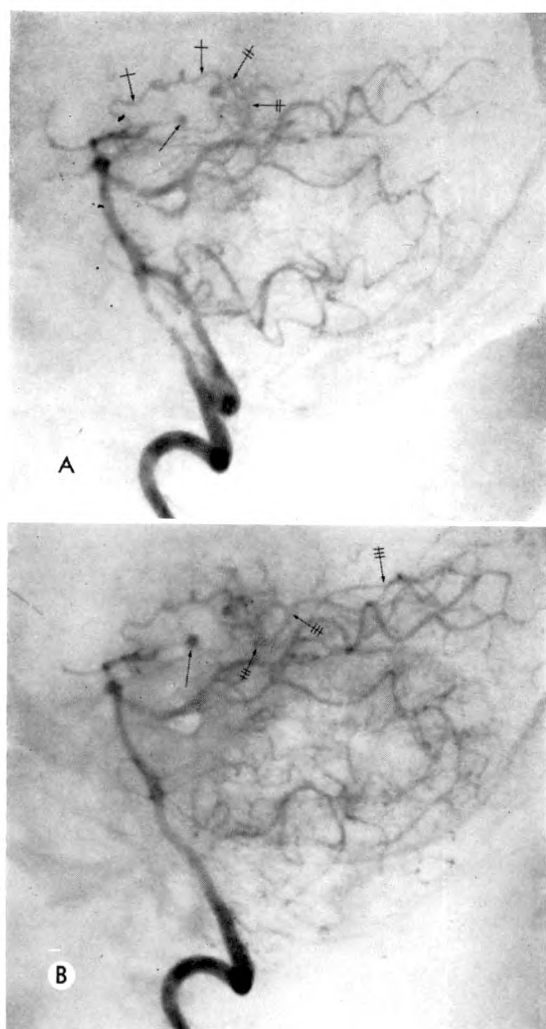


FIG. 1. Case 1. Second order subtraction film from a vertebral angiogram with the patient obliqued to prevent superimposition of the two posterior cerebral arteries. (A) A small amount of contrast material has entered the aneurysm ( $\rightarrow$ ). The medial posterior choroidal artery is filled ( $\leftrightarrow$ ) and there is beginning retrograde filling of the lateral posterior choroidal artery ( $\#$ ). (B) Study made 1.0 second later in the series. The aneurysm is better filled ( $\rightarrow$ ). Contrast material now outlines the distal portion of the posterior cerebral artery ( $\#$ ).

On December 31, 1965, he had sudden onset of excruciating headache with concomitant left-sided weakness and hemiparesis starting in his face and progressing to involve the left half of the body. The numbness and weakness lasted about 20 minutes, the headache for several hours.

The patient had a second similar episode on January 14, 1966, and a third episode in the evening of January 23, 1966. With the latter episode, he noted a transient left homonymous hemianopsia.

Lumbar puncture at another hospital showed grossly bloody xanthochromic spinal fluid and an elevated spinal fluid pressure. He was transferred to Wadsworth V.A. Hospital where physical examination showed a drowsy 23 year old Negro male, lying quietly in bed. Rectal temperature was  $100.4^{\circ}\text{F}$ ., blood pressure 130/70 and the pulse rate 80. Memory was clouded and often inconsistent but he was oriented in all spheres and calculated well with prodding. His speech was normal.

Cranial nerve examination showed no significant abnormalities and the motor examination was normal except for slowing in the performance of rapid alternating movements. Sensory testing was normal. Reflexes were hypoactive but symmetric. The plantar responses were flexor.

Bilateral carotid angiography and vertebral angiography were performed on January 27, 1966, showing an aneurysm arising from the right posterior cerebral artery (Fig. 1, A and B). An intracarotid injection of 10 cc. sodium amytal showed that the patient had left-sided cerebral dominance.

During hospitalization, his sensorium gradually improved and his nuchal rigidity diminished. On February 3, 1966, a right temporal craniotomy was performed under general anesthesia. The aneurysm was about 9 mm. in diameter and surrounded by old blood and yellowed cortex. The posterior cerebral artery was successfully clipped, both proximally and distally to the aneurysm. The collateral vessels visible on the angiogram were identified and preserved. The patient had an uneventful postoperative course, his sensorium continued to clear and he has remained totally intact neurologically.

Presumably, this patient had experienced multiple episodes of small intracranial bleeding which caused prolonged spasm of the right internal carotid artery. The aneurysm was situated on the posterior cerebral artery between the origin of the medial posterior choroidal artery and the lateral posterior choroidal artery. The relative anoxemia of the brain allowed dilatation of collateral vessels such that even with intense spasm, sufficient to cause total occlusion

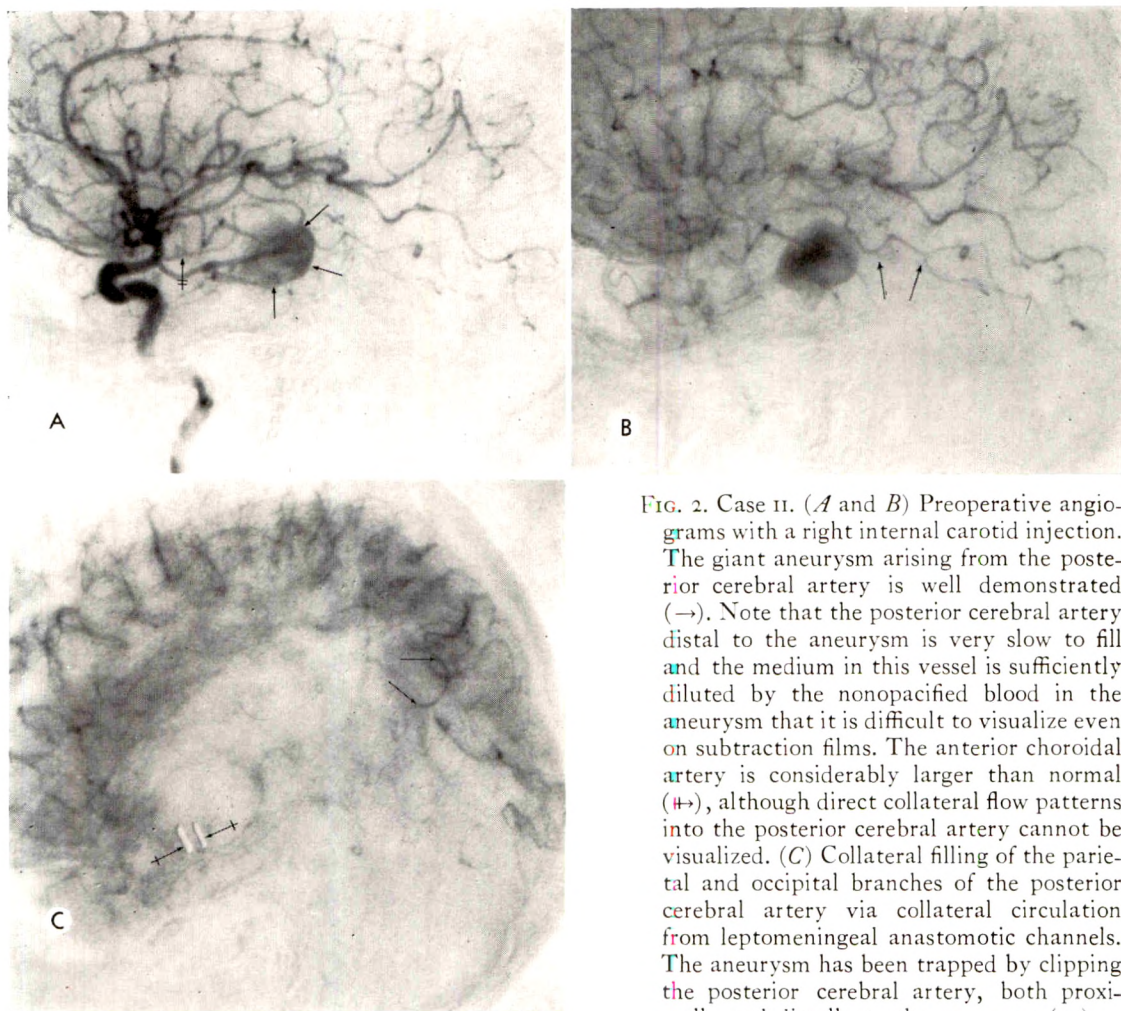


FIG. 2. Case II. (A and B) Preoperative angiograms with a right internal carotid injection. The giant aneurysm arising from the posterior cerebral artery is well demonstrated ( $\rightarrow$ ). Note that the posterior cerebral artery distal to the aneurysm is very slow to fill and the medium in this vessel is sufficiently diluted by the nonopacified blood in the aneurysm that it is difficult to visualize even on subtraction films. The anterior choroidal artery is considerably larger than normal ( $\leftrightarrow$ ), although direct collateral flow patterns into the posterior cerebral artery cannot be visualized. (C) Collateral filling of the parietal and occipital branches of the posterior cerebral artery via collateral circulation from leptomeningeal anastomotic channels. The aneurysm has been trapped by clipping the posterior cerebral artery, both proximally and distally to the aneurysm ( $\leftrightarrow$ ).

of the posterior cerebral artery at the level of the aneurysm, the distal posterior cerebral artery remained patent. Postoperative angiography confirmed the patency of the collateral vessels and the distal posterior cerebral artery.

**CASE II.** A 20 year old, right-handed white male was well until 5 days prior to his admission when he developed sudden, severe left temporal headache, radiating behind the left eye, followed by nausea and vomiting. The temperature was  $37.6^{\circ}\text{C}$ . and because of a current influenza epidemic, the patient was initially thought to be suffering from this disease. He improved on symptomatic care until early on the day of admission when he had a second episode of severe headache, again sudden in onset, but this time bifrontal. This was accompanied by neck stiffness, a pulse rate of 68 and a blood

pressure of 102/62. Lumbar puncture was performed at this time and revealed grossly bloody spinal fluid. The general medical history was totally unremarkable.

On physical examination, the patient appeared to be in moderately severe discomfort due to headache and photophobia. His neck was rigid. He appeared alert and fully oriented and there was no evidence of any intellectual deficit. His cranial nerve examination disclosed a mild anisocoria as the only positive finding, the left pupil being minimally larger than the right. Both pupils reacted well to light. There was no ptosis. Motor examination showed normal strength, tone and coordination of the extremities. The reflexes were symmetric and no pathologic reflexes were elicited.

Preoperative angiograms (Fig. 2, A and B) were obtained, showing a large aneurysm aris-



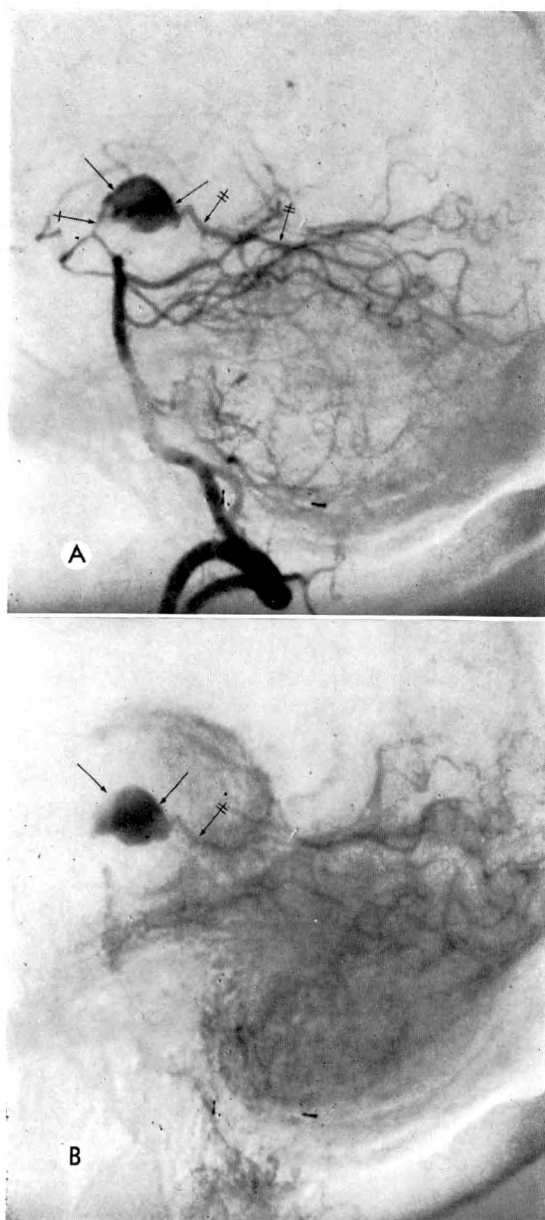


FIG. 3. Case III. (A) Note the large size of the aneurysm which is only partially filled (→). The proximal posterior cerebral artery (↔) is slender and displaced superiorly by the nonopacified portion of the artery. The posterior cerebral artery distal to the aneurysm (↔) is of normal caliber and well filled. (B) Study shows the very slow emptying of the posterior cerebral artery (↔) while the contrast material has entered into the capillary phase in other regions. The aneurysm (→) is filled to a greater extent.

edema did not appear to respond well to osmotic diuretics. After elevating the temporal lobe, the aneurysm and its feeding artery were visualized. Two silver clips were placed across the branch of the posterior cerebral artery which entered the aneurysm. The aneurysm was then seen to collapse. At the time of closure, there was severe edema so that the bone flap was left out and the dura could not be reapproximated.

Postoperatively, the patient was quite somnolent, totally aphasic and had a right hemiplegia. Purposeful movements of the left extremities became apparent in the early postoperative period. His condition improved slowly to the point that he was able to assist actively in feeding himself, using his left extremity.

The angiogram showed a giant type of aneurysm which filled the posterior cerebral arteries very slowly distally to the aneurysm. Some collateral circulation may well have been taking place as evident by the large size of the anterior choroidal artery on the preoperative angiogram. On the postoperative study (Fig. 2C) he demonstrated retrograde filling of the occipital branches of the posterior cerebral artery via leptomeningeal collateral circulation.

ing from the proximal left posterior cerebral artery. Approximately 3 hours after an uneventful arteriographic study, the patient had a severe exacerbation of headache, associated with pallor and bradycardia with a pulse rate of 36. However, he was able to speak and had use of all of his extremities. His sensorium was somewhat dull compared to his state immediately prior to this last episode. The patient was immediately taken to the operating room and a left temporal craniotomy was performed. The hemisphere was extremely edematous and the

CASE III. On January 8, 1965, a 20 year old Negro man was admitted complaining of severe posterior parieto-occipital headaches, worse on the right than on the left. For 3 months prior to admission, he began to have headaches each morning upon arising, which would gradually wear off during the course of the day. Three weeks prior to admission, he noticed drooping of his right eyelid and diplopia which became progressively worse. On admission, the right eye was completely shut. He has had two episodes of unsteady gait which were transient.

On neurologic examination, he had a complete left third nerve palsy and a left central facial weakness. Motor examination demonstrated very slight weakness of the left arm and reflexes tended to be more brisk on the left, although the plantar responses were flexor bilaterally. On January 14, 1965, bilateral internal carotid angiography and vertebral angiography were performed (Fig. 3, *A* and *B*).

On January 21, 1965, a right temporoparietal craniotomy with partial right temporal lobectomy was performed. An aneurysm was encountered, measuring approximately 2×4 cm. in diameter, indenting and adherent to the brain stem. An effort was made to reduce the size of the aneurysm. Considerable bleeding occurred as the thrombus was being removed from the aneurysm. This bleeding was eventually controlled by packing with Surgicel. The patient remained somnolent following surgery and a shunting procedure was performed to relieve his hydrocephalus. This required revision at a third operation on February 8, 1965. On February 11, 1965, he experienced a generalized seizure and became unresponsive. He expired on March 3, 1965. Autopsy showed massive subarachnoid bleeding at the site of the aneurysm.

This patient showed no evidence of collateral circulation on the preoperative angiogram. The posterior cerebral artery distal to the aneurysm filled promptly with contrast material of good density. The aneurysm was quite large, filled well and was very slow to empty. Had adequate collateral circulation been demonstrated angiographically, a "trapping procedure" may have been more attractive to control the bleeding at the first operation.

#### DISCUSSION

In any given group of patients showing evidence of cerebral infarction, a small percentage are found in whom the physical findings and spinal fluid examinations suggest a ruptured intracranial aneurysm. Angiography may demonstrate only an occluded vessel and an aneurysm is not visualized. Similarly, some patients with symptoms of cerebral infarction show, at angiography, an aneurysm with surrounding vascular spasm. The spasm may be exceedingly severe; however, some contrast material almost always flows through the

vessels. Rarely is it possible to demonstrate an aneurysm filling while the vessel distal to the aneurysm is totally occluded. Case 1 clearly showed angiographic occlusion of the posterior cerebral artery for a distance of at least 5 mm. distal to the aneurysm. Segmental spasm must have developed sufficiently slowly to allow collateral circulation to develop between the medial posterior choroidal artery and the lateral posterior choroidal artery so that cerebral infarction did not take place in the distribution of the posterior cerebral artery.

The 3 patients are reported to show strikingly different postoperative courses. We do not mean to imply that collateral flow patterns are the most important factor in prognosticating the postoperative course. Size and location of the aneurysm, extracerebral as well as intracerebral bleeding, vascular spasm and cerebral edema are but a few of the factors deciding the eventual fate of the patient. However, the radiologist plays a vital role in demonstrating the fine vascular detail. By alerting the neurosurgeon to leptomeningeal collateral flow patterns, care may be taken at surgery to preserve these vessels.

Roentgenograms taken at the rate of 3 or 4 per second over a period of 5 seconds are generally required to demonstrate the retrograde flow of contrast material in an artery. By placing a catheter either in the internal carotid artery or in the vertebral artery, much better filling of the intracranial vessels can be obtained. The improved filling without an overlap of external carotid circulation is exceedingly helpful in detecting differences in circulation time.

#### SUMMARY

Aneurysms may produce sufficient spasm to cause angiographic nonfilling of an intracranial artery. We believe this nonfilling to be due to thrombosis but the actual cause of the nonfilling is difficult to prove without histologic examination. If the spasm proceeds slowly enough, collateral flow to the ischemic area may develop from the



leptomeningeal vessel to a sufficient degree to prevent cerebral infarction.

The neuroradiologist plays a vital role in demonstrating these fine collateral flow patterns and alerting the neurosurgeon to their presence.

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## HEART FAILURE IN INFANCY DUE TO ARTERIOVENOUS MALFORMATION OF THE VEIN OF GALEN\*

### REPORT OF A CASE

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**A**N ANEURYSM of the vein of Galen consists of an arteriovenous malformation involving the carotid and/or the vertebrobasilar circulation in direct communication with the great cerebral vein. An arterial branch may enter the vein directly, or there may be a complex racemose network of noncapillary vessels interposed between the artery and vein. Nearly 50 cases of this congenital malformation have been reported. A recent review summarizes many of these.<sup>6</sup> The age range at which the aneurysm first becomes symptomatic is from birth to adulthood, and the earliest clinical manifestations include focal pressure signs, obstructive hydrocephalus, evidence of intracranial bleeding, and a high output type of heart failure.

Because the first sign of this vascular anomaly may be cyanosis and congestive heart failure in newborns and infants and because such a syndrome has seldom been recognized prior to death, a case is presented in which the diagnosis was made ante mortem. Surgical correction was attempted but failed. The surgical and post-mortem findings corroborated the angiographic findings.

#### REPORT OF A CASE

A 5½ month old white male infant was admitted to The New York Hospital on October 19, 1963, because of uncontrolled congestive heart failure and an enlarged head. He was born after a normal pregnancy, to a healthy mother of 3 children. Delivery was normal and the child did well until 2½ months of age when an elective inguinal herniorrhaphy was per-

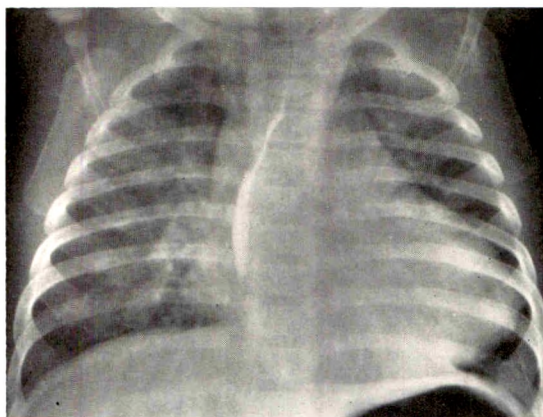


FIG. 1. Frontal esophagram showing massive enlargement of the heart and congestion of the lungs.

formed without complication. At 3 months, he had a generalized seizure with cyanosis and was admitted to another hospital where a heart murmur, cardiomegaly, and increased pulmonary vascular markings were noted. Cardiac catheterization was carried out at another hospital at 4½ months of age and congenital heart disease was diagnosed. At 5 months the child had blue swellings of both eyelids which became more prominent. He subsequently had another generalized seizure with cyanosis, at which time congestive heart failure was recognized and digitalis prescribed. His head was abnormally large and the scalp veins were prominent.

On admission to The New York Hospital, he had a symmetrically enlarged head (45 cm. in circumference), a full and strongly pulsatile anterior fontanelle, and dilated veins over the scalp and both eyelids. A loud systolic bruit was heard over the entire cranium. A chest roentgenogram (Fig. 1) demonstrated massive en-

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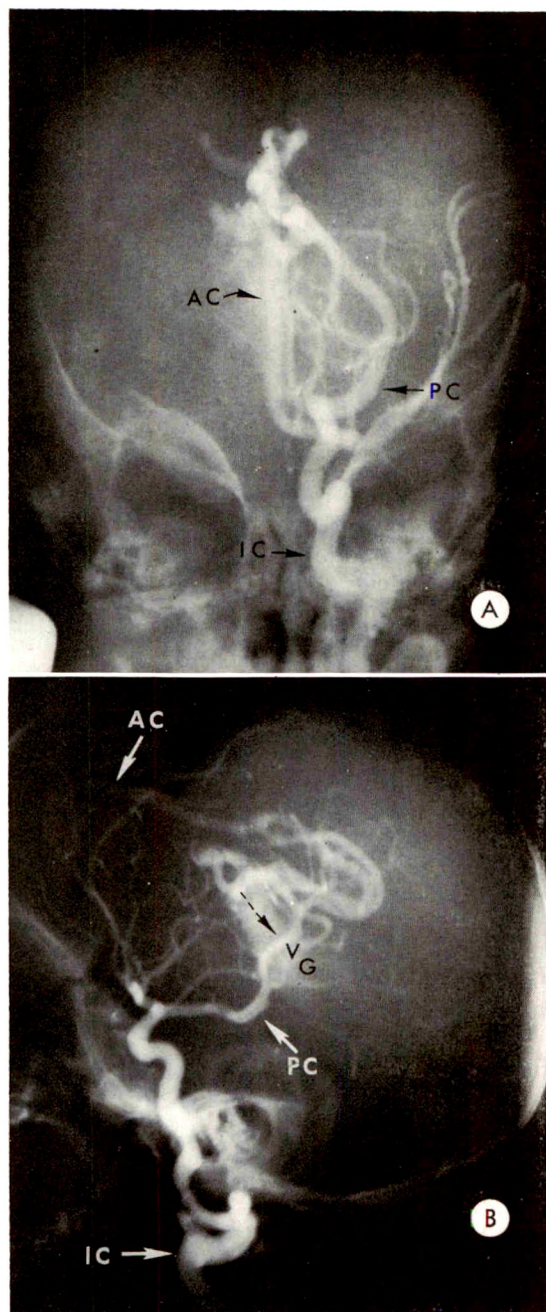


FIG. 2. (A) Frontal and (B) lateral left carotid arteriograms showing enlarged cervical portion of internal carotid artery (IC). Note the enlarged anterior and posterior cerebral arteries (AC, PC) in comparison with the normal sized middle cerebral artery. At 0.33 second, a small amount of contrast medium is shunted into the vein of Galen (VG) via a network of malformed arteries.

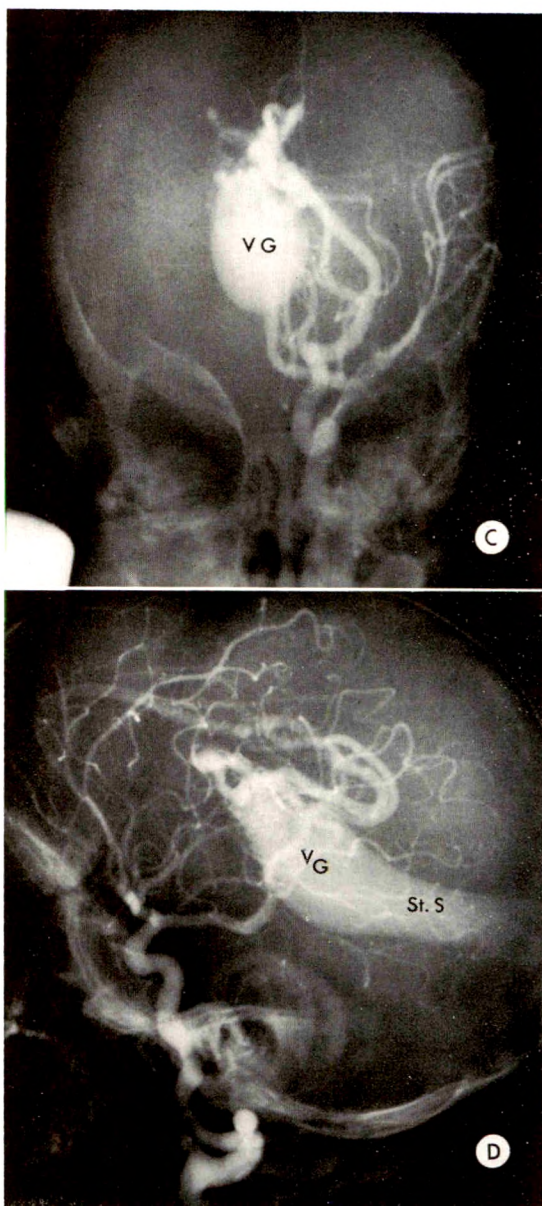


FIG. 2. (C) Frontal and (D) lateral views. Still early in the arterial phase, contrast medium is rapidly filling a huge aneurysm of the vein of Galen (VG) and straight sinus (St. S).

largement of the heart. Left carotid and right vertebral arteriograms demonstrated a massively dilated vein of Galen which was continuous with the left anterior cerebral and both posterior cerebral arteries (Fig. 2, A-F). A ventriculogram showed the aneurysm of the vein of Galen outlined by air in the posterior third ventricle as the cause of obstructive hydro-



cephalus (Fig. 3). The clinical diagnosis on the basis of roentgenographic studies was an arteriovenous malformation of the vein of Galen.

Because the anomaly was causing obstructive hydrocephalus and because cardiac compensation could not be maintained, a decision to

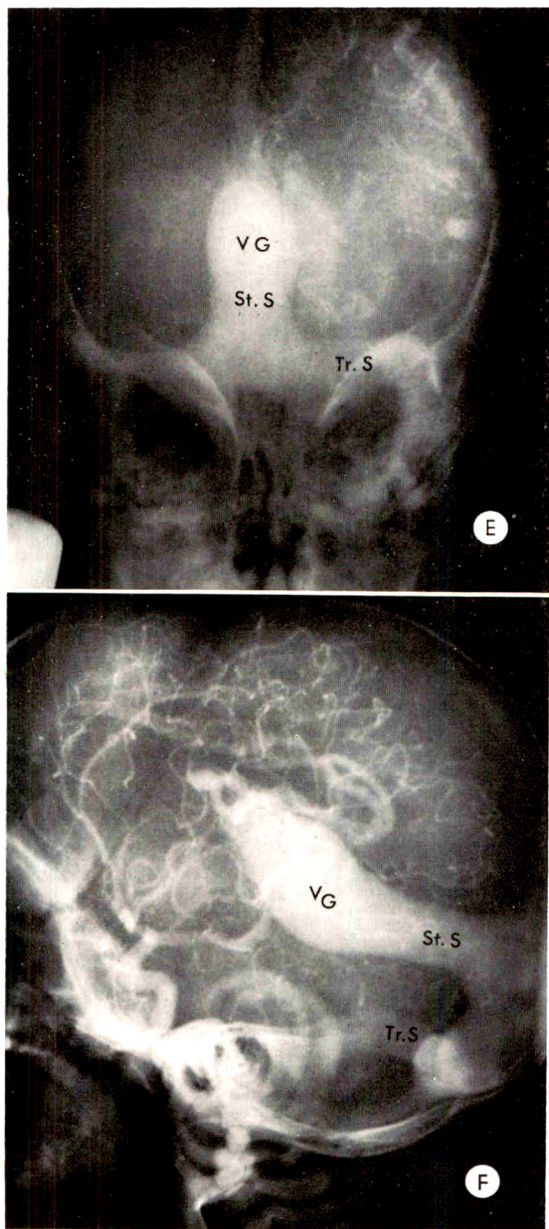


FIG. 2. (E) Frontal and (F) lateral views. Late arterial and venous phases showing the aneurysm of the vein of Galen (VG), markedly dilated straight sinus (St. S), torcular Herophili, and dilated transverse (Tr. S) and sigmoid sinuses.

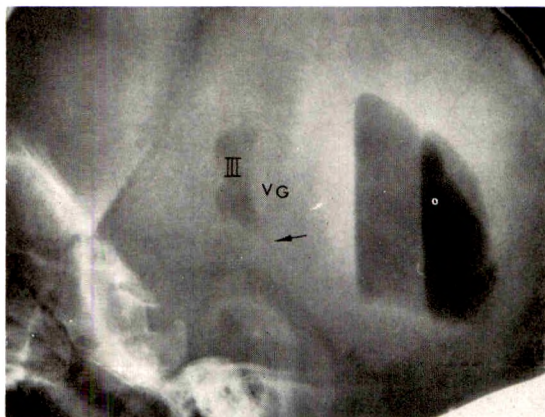


FIG. 3. Lateral ventriculogram demonstrating internal hydrocephalus with obstruction at aqueduct (arrow). Anterior portion of the aneurysm (VG) is clearly outlined by the air in the posterior third ventricle (III).

operate was made. A 3 cm. aneurysm of the vein of Galen was exposed via a left occipital craniotomy. It lay in the hiatus between the brain stem and the edge of the tentorium. Approximately 10 to 15 small arteries entered the aneurysm on either side. The arterial supply and the enlarged straight sinus were divided, and the aneurysm was excised without difficulty. However, at the conclusion of the operation, the patient's heart developed ventricular fibrillation and he died.

At postmortem examination, the heart and lungs together weighed 220 gm. (expected weight 107 gm.). All of the chambers of the heart were dilated; the myocardium of the left and right ventricles was 7 and 3 mm. in thickness, respectively. All other cardiac structures were normal; no anomaly was present. The head was enlarged and 44 cm. in circumference (expected circumference for age is 42 cm.). The cerebral hemispheres were separated and the intervening space was filled with approximately 25 cc. of clotted blood. There was marked engorgement of the subarachnoid vessels at the base of the brain. Numerous severed vessels were clamped by metal clips. The quadrigeminal plate was markedly flattened. On section, the ventricles were moderately dilated and there was atrophy of the adjacent white matter. Within the cerebral parenchyma a few millimeters lateral to the lateral ventricle, there were several large, tortuous arteries, which often bulged into the adjacent ventricle (Fig. 4). The frontal lobes contained bilateral areas of



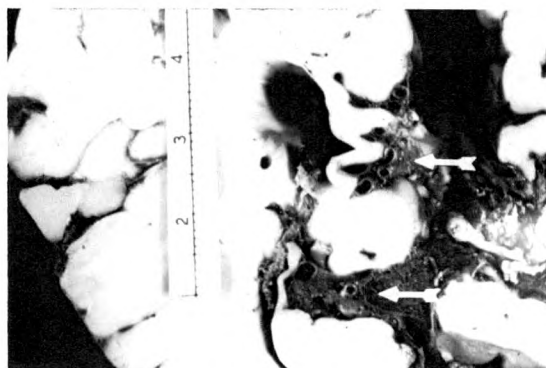


FIG. 4. Photograph of coronal section of the mid-portion of the left cerebral hemisphere showing moderate dilatation of the left lateral ventricle and numerous anomalous tortuous, dilated arteries that supplied the aneurysm (arrow).

softening and cavitation. Microscopically, in numerous sections from the frontal lobes and cerebellum, the parenchyma and meninges contained numerous clusters of small abnormal vessels. The walls of many intracerebral vessels were focally calcified.

There was hyperemia of the lungs, liver, kidneys and spleen. Fresh blood was noted in the upper gastrointestinal tract with occasional small areas of hemorrhage in the mucosa. All other structures were normal.

#### COMMENT

Cardiovascular dysfunction has been recognized as a complication of intracranial arteriovenous malformations since Ray's<sup>13</sup> extensive review in 1941. The first 2 cases of heart failure occurring secondary to an aneurysm of the vein of Galen were reported by Clément *et al.*<sup>2</sup> in 1954. Since then no less than 17 cases of heart failure secondary to this lesion have been reported (Table 1).<sup>1,3-8,11</sup> In only 4 of these was the diagnosis made prior to death, and then only by cerebral arteriography. Two of the 17 patients were operated upon in attempts to correct the arteriovenous fistula.<sup>6,7</sup> Both survived surgery; 1 died later in a decerebrate state and a follow-up report of the second is not available. One patient had multiple shunting procedures performed for hydrocephalus, and the correct diagnosis was made only at autopsy.<sup>5</sup> Examina-

tion of the brain in each of the 13 patients, in whom diagnosis was made at autopsy, showed a large aneurysmal dilatation of the vein of Galen that was supplied by varying branches of the cerebral arteries. Other extracranial congenital anomalies were rare; 2 patients had congenital heart disease.<sup>7,8</sup>

Fourteen of the 17 patients died in congestive heart failure secondary to the arteriovenous malformation of the vein of Galen (Table 1). In 14, the onset of failure occurred in the first week of life, but 3 did not develop failure until 1, 4, and 7 months of age, respectively. Our patient was asymptomatic until the age of 3 months. In most instances, a diagnosis of congenital heart disease was made prior to cerebral arteriography or autopsy. Cyanosis and heart murmurs were frequent, and the cardiac failure was often difficult to control medically (Table 1).

The mechanism of heart failure secondary to intracranial arteriovenous fistulas has been discussed in the past.<sup>9</sup> This type of cardiovascular dysfunction has been referred to as hyperkinetic heart failure. The low peripheral resistance resulting from a direct communication between arterial and venous circulations causes both increased cardiac output and a compensatory hypervolemia. The mechanisms by which these changes take place are poorly understood. Because the venous return to the heart is greatly increased without a concomitant reduction in pulmonary vascular resistance, right ventricular heart failure may supervene. Walker *et al.*<sup>15</sup> proposed that the cyanosis, often observed in infants with large arteriovenous fistulas, results from a massive right-to-left shunting of blood through the foramen ovale, secondary to an elevated right atrial pressure.

It has been recognized that heart failure secondary to intracranial arteriovenous malformations appears most frequently in the neonatal period. Gold *et al.*<sup>6</sup> divided patients with vein of Galen malformations into 3 groups with respect to the age at which symptoms first appeared. They are:

TABLE I

SUMMARY OF CASES OF CONGESTIVE HEART FAILURE SECONDARY TO ARTERIOVENOUS MALFORMATION OF THE VEIN OF GALEN

Case	Source	Sex	Age at Onset of CHF	Cardiovascular Manifestations	Other Findings	Diagnosis	Outcome
1	Clément <i>et al.</i> <sup>2</sup> (1954)	M	4 weeks	Cyanosis; peripheral edema; pulmonary edema	Tense fontanelle	PM	Died, CHF; age 38 days
2		F	7½ months	Cardiomegaly; cyanosis	Hydrocephalus	A	Died, CHF; age 8 months
3	Pollock and Laslett <sup>11</sup> (1958)	M	2 days	Cardiomegaly; tachycardia; tachypnea	Hydrocephalus	PM	Died, CHF; age 83 hours
4	Corrin <sup>3</sup> (1959)	F	2 days	Cardiomegaly; dyspnea		PM	Died, CHF; age 4 days
5	Claireaux and Newman <sup>1</sup> (1960)	M	16 hours	Cyanosis; systolic murmur; hepatomegaly	Supernumerary digits; normal electroencephalogram	PM	Died, CHF; age 4 days
6		M	1 week	Cyanosis; cardiomegaly; peripheral edema		PM	Died, CHF; age 16 days
7	Gagnon and Boileau <sup>4</sup> (1960)	M	Birth	Cyanosis; cardiomegaly; pulmonary edema	Tense fontanelle	PM	Died, CHF; age 2 days
8	Glatt and Rowe <sup>5</sup> (1960)	M	3½ hours	Cardiomegaly; systolic murmur; tachypnea	Hydrocephalus	PM	Cerebrospinal fluid shunt performed; died, meningitis at age 5 months
9		M	32 hours	Cyanosis; gallop rhythm; hepatomegaly		PM	Died, CHF; age 70 hours
10	Hirano and Solomon <sup>8</sup> (1960)	M	Birth	Cyanosis; cardiomegaly; hepatomegaly		PM	Died, CHF; age 7 weeks
11	Gomez <i>et al.</i> <sup>7</sup> (1963)	M	4 months	Cardiomegaly; hepatomegaly; pulmonary edema	Cranial bruit	A	Died, CHF; age 6 months
12		M	12 hours	Cyanosis; cardiomegaly; systolic murmur		PM	Died, CHF; age 36 hours
13		M	Birth	Cyanosis; cardiomegaly; hepatomegaly		PM	Died, CHF; age 2 days
14		M	Birth	Cyanosis; cardiomegaly; dyspnea		PM	Died, CHF; age 2 days
15		F	3 days	Cardiomegaly; systolic murmur; hepatomegaly	Cranial bruit	A	Survived following right carotid ligation
16	Gold <i>et al.</i> <sup>6</sup> (1964)	M	Birth	Cyanosis; cardiomegaly; hepatomegaly	Cervical cystic hygroma	PM	Died, CHF; age 2 days
17		F	Birth	Cardiomegaly; systolic murmur; hepatomegaly	Hydrocephalus; cranial bruit	A	Died in decerebrate state 2 years following clipping of aneurysm
18	Present case	M	3 months	Cardiomegaly; systolic murmur	Hydrocephalus; cranial bruit	A	Died at operation

A=cerebral arteriography.  
 PM=post mortem.  
 CHF=congestive heart failure.



the neonatal period, in which heart failure predominates; infancy, in which convulsions and hydrocephalus appear first; and older children and adults, in whom headache and focal signs predominate. Most commonly, infants with this anomaly present with convulsions and hydrocephalus. However, as illustrated by our infant patient and Case 1 of Gomez *et al.*,<sup>7</sup> when congestive failure precedes hydrocephalus in the natural history of this disorder, diagnosis is delayed.

The vein of Galen malformations should be included in the differential diagnosis of heart failure of obscure cause in the neonatal and infant periods. The syndrome of congestive heart failure, hydrocephalus, and a cranial bruit in an infant should suggest the diagnosis of an aneurysm of the vein of Galen. Carotid arteriography usually demonstrates the lesion. Vertebral arteriography should always be performed since the posterior cerebral arteries are the most common supplying vessels, and bilateral carotid arteriograms may not demonstrate filling of the aneurysm.<sup>13</sup> Ventriculograms show a filling defect in the third ventricle, and, if hydrocephalus is present, there is symmetric enlargement of the third and lateral ventricles.

The surgical treatment has been reviewed by Pool and Potts,<sup>12</sup> who favor a staged procedure. First, the nutrient artery or arteries are clipped on 1 side only. This may be sufficient to relieve symptoms and prevent subsequent complications. A second operation is usually necessary, at which time the nutrient arteries on the other side are divided, with or without resection of the aneurysm. Racemose types of arteriovenous malformations are usually not amenable to correction.<sup>10</sup>

Most of the surgical experience with vein of Galen malformations has been in patients who presented with symptoms other than cardiovascular.<sup>12</sup> However, closure of the arteriovenous communication in patients not in heart failure may precipitate acute cardiac decompensation due to sud-

den return of a large blood volume to effective circulation. Hence, the blood volume and venous pressure must be carefully measured pre- and postoperatively in all patients in whom correction is attempted.

#### SUMMARY AND CONCLUSIONS

An infant with heart failure secondary to an arteriovenous malformation of the vein of Galen is described. The onset of congestive failure began at the age of 3 months. The diagnosis of an arteriovenous fistula was established by cerebral arteriography at the age of 6 months. The diagnosis was suggested by the presence of hydrocephalus and a cranial bruit. Surgical correction was attempted at 6½ months of age, but the infant died of ventricular fibrillation.

The literature on vein of Galen malformations is reviewed with respect to those patients presenting with heart failure. Seventeen patients with heart failure secondary to this lesion have been reported; 13 were neonates and 3 were infants of 1, 4, and 7 months of age. Fourteen patients died in heart failure, 2 died following surgery, and only 1 survived.

The mechanism of heart failure in intracranial arteriovenous malformations is poorly understood, but involves increased cardiac output and hypervolemia. The syndrome of congestive heart failure, hydrocephalus, and cranial bruit should suggest the diagnosis of an aneurysm of the vein of Galen. This diagnosis should always be considered in neonates and infants with heart failure of obscure etiology.

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## ANGIOGRAPHY OF THE POSTERIOR CERVICOCRANIAL CIRCULATION\*

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VARIOUS angiographic techniques have been employed in the study of patients with insufficiency of the vertebrobasilar system or with lesions in the posterior cranial fossa. The purpose of the authors, however, is not to evaluate these methods but rather to report modifications in our own technique of percutaneous infraclavicular catheterization of the subclavian artery.<sup>6,7</sup> These improvements have enabled us to delineate more precisely lesions which can be treated surgically, and should prove beneficial to others. The method was used initially in the study of patients with symptoms of cerebral ischemia presumed to be due to vascular occlusive disease. However, it has been found to be extremely useful for visualization of other abnormalities such as saccular and fusiform aneurysms, arteriovenous malformations, and space-occupying lesions in the posterior cranial fossa.

Many physicians recognize the need for employing angiography in the diagnosis and localization of lesions in the posterior cerebral circulation, but most insist that satisfactory delineation can be achieved only by direct vertebral injection, which is known to be technically difficult and potentially dangerous.<sup>8</sup> We have been able with less risk to adequately opacify the vessels in the posterior fossa indirectly by means of catheterization of the subclavian arteries.

Anatomic and roentgenologic studies of the vertebral arteries have revealed a high incidence of morphologic variations such as unilateral or bilateral hypoplasia and tortuosity in the cervical segment. These abnormalities may increase the technical difficulties in performing direct vertebral puncture, and for this reason indirect injection is preferred. Furthermore, a needle inserted directly into one of the vertebral arteries prohibits freedom of movement of

the head and neck so that certain essential roentgenographic projections cannot be used.

From July, 1962, to September, 1965, subclavian catheterization was employed in 846 patients (580 males and 266 females) between the ages of 4 and 91 years admitted to The Methodist Hospital, Houston, Texas. The prearteriographic diagnosis in the majority of these patients was cerebrovascular insufficiency due to atherosclerotic occlusive disease, but 39 patients were admitted with diagnoses of other types of lesions in the posterior cranial fossa or posterior cervicocranial circulation, including 15 aneurysms, 6 arteriovenous malformations, 9 tumors of the posterior fossa, and 9 cases of extrinsic compression of the vertebral artery.

The procedure was performed using general anesthesia in 583 patients (81 per cent) and local anesthesia in 135 (19 per cent). It has been our observation that most patients will tolerate the injection of a large amount of contrast material much better when under general anesthesia. For this reason and for the convenience of the arteriographer, general anesthesia has been used in the majority of these cases since early in 1964.

Meglumine iothalamate 60 per cent (Conray, Mallinckrodt) was employed in all cases, and the injections were made manually. The amount of contrast material used in a single injection was from 5 to 30 ml., and the total amount injected was between 50 and 200 ml. In this series of patients, there were 785 bilateral and 61 unilateral subclavian catheterizations, making a total of 1,631.

### MATERIAL AND METHOD

In order to carry out this procedure, the

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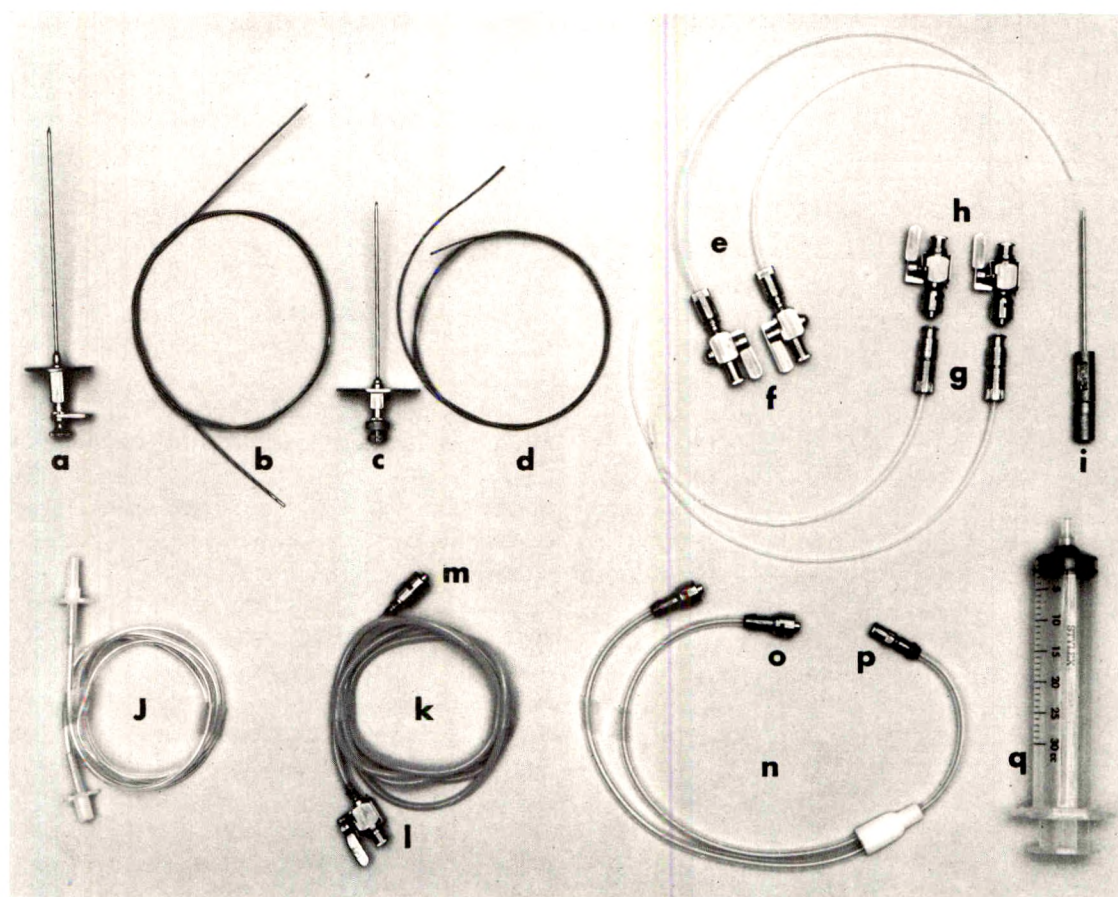


FIG. 1. (a-q) Equipment used in direct percutaneous infraclavicular catheterization of the subclavian artery. (For details see Appendix.)

following equipment, prepared as described, is required (Fig. 1, a-q).\*

1. A Cournand-Grino needle (Fig. 1a), XT487LNR, 17 gauge,  $3\frac{1}{2}$  inches long, or a Seldinger needle (Fig. 1c), PE 160.

2. A flexible metallic guide, 19 gauge (Fig. 1b), to fit the Cournand-Grino needle. A 20 gauge metallic guide (Fig. 1d) is used with the Seldinger needle. Both of these guides should be 80 cm. in length with an 8 to 10 cm. straight flexible tip, which is easy to thread into a tortuous artery.

3. Polyethylene tubing (Fig. 1e), PE 190 or PE 200 in caliber, 20 to 25 cm. in length. It is very important that the tip of the catheter be properly prepared in order to facilitate its insertion over the metallic guide and through the skin and wall of the

artery. The heat from a lighted cigarette is used to flare out the end of the catheter to be fitted into the tubing adapter. The other end is adjusted to the diameter of the metallic guide by gently stretching a segment and heating it with steam. The metallic guide is inserted into the tubing, which is then cut with a razor blade at the point where advance of the tip of the metallic guide is impeded by the narrowed lumen (Fig. 2).

When polyethylene tubing of larger caliber (PE 200) is used, to facilitate manual injection, a hole is made 1 to 1.5 cm. from the tip of the catheter using a punching instrument (Fig. 1i). Tubing of this caliber is used mostly for injection of more concentrated contrast material (Angio-Conray 80 per cent, Mallinckrodt) into the aortic arch.

\*A list of manufacturers of this equipment with numbers corresponding to those used in the paper may be found in the Appendix.



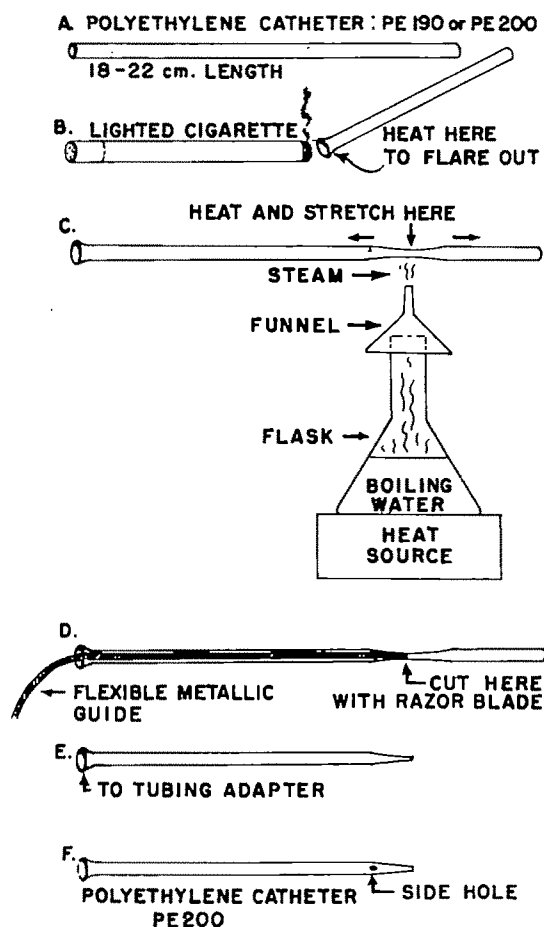


FIG. 2. Steps in the preparation of polyethylene catheter for arterial catheterization.

4. A tubing adapter (Fig. 1g), A-1025, size C, is fitted to the flared end of the polyethylene tube. Recently, we have been employing a removable fitting (Fig. 1f), #BR 80 or BR 90. With this device, the tubing adapter and the one-way stopcock may be connected in one piece. The tubing is then connected to a 30 ml. syringe by means of a flexible plastic connecting tube 50 cm. in length (Fig. 1j), which permits the operator to remain behind a shield out of the field of radiation. For injection of the aortic arch, the plastic tube is cut at both ends and one end inserted into a one-way stopcock, B.D. MSo<sub>4</sub>-T (Fig. 1l), and the other end is inserted into an adapter, B.D. 608/L (Fig. 1m). The adapter and stopcock are secured by winding a piece of wire

around the tubing to permit high pressure during injection.

5. A one-way stopcock, B.D. MSo<sub>9</sub> (Fig. 1h), is fitted to the tubing adapter of the polyethylene catheter, which permits the system to be opened or closed as desired.

6. A Y-shaped connecting tube (Fig. 1n) for simultaneous bilateral injection of the subclavian arteries. This bifurcated tube can be obtained with the set for hypodermoclysis. The tube is cut 12 cm. from the bifurcation and an adapter, B.D. H/468L (Fig. 1o), fitted at each end for connection to the adapters on the polyethylene tube. An adapter, B.D. L/606 (Fig. 1p), is placed on the single stem of the Y-shaped tube so that it may be connected to the 30 ml. syringe (Fig. 1q) by attaching it to the long plastic tube previously described. A Y-connection adapter, B.D. 439A, female Luer-Lok to two male Luer-Loks may be employed for the same purpose.

7. Normal saline solution, 300 to 400 ml., containing 50 mg. of sodium heparin per 100 ml., is needed for perfusion of the tubing at regular intervals to avoid clotting.

8. The polyethylene tubing, prepared in advance, and the Y-shaped connecting tube are kept sterile in colorless Zephiran solution (Winthrop Laboratories) and washed with normal saline solution before being used.

#### TECHNIQUE

The patient is placed on the table in a supine position with his neck in neutral position or slightly hyperextended by a roll under the shoulders, making it easier to palpate the subclavian artery above the clavicle. This is important for determining the approximate position of the vessel before advancing the needle from the site of infraclavicular puncture.

The site of needle puncture in the infraclavicular region is at the junction of the inner and middle third of the clavicle. Although the point at which the subclavian artery crosses the inferior border of the clavicle varies slightly from one patient to

another, a review of many angiograms has shown that it is usually between 5.5 and 6.5 cm. from the superior angle of the medial end of the clavicle. In some patients it crosses at a point slightly more lateral but seldom more medial.

With the fingers of one hand, the subclavian arterial pulse is palpated in the supraclavicular fossa, and, with the free hand, the double needle is inserted beneath the clavicle and advanced superiorly, medially, and posteriorly toward the palpable pulsation. The artery at this point is superficial and lies over the first rib. The branches of the brachial plexus are above and behind the vessel. When the tip makes contact with the first rib, the needle should not be inserted further. At this depth one should be able to puncture the artery without injuring the pleura and lung. Palpation and puncture of the artery are often difficult if the systolic blood pressure is less than 100 mm./Hg. Under such circumstances, the anesthetist can assist by administering vasopressor drugs.

Once the tip of the double needle is within the lumen of the artery, the sharp inner needle is withdrawn and the outer sheath adjusted by gently advancing or withdrawing until satisfactory pulsatile blood flow is obtained (Fig. 3*a*). The flexible end of the metallic guide is then introduced through the outer needle until 4 or 5 cm. has been inserted into the arterial lumen (Fig. 3*b*). The guide must be advanced with care. If resistance is encountered, it may be due to the position of the needle with respect to the wall of the artery, and a slight change in the direction of the needle may facilitate insertion. On occasion, the tip of the guide may enter the thyrocervical trunk or the vertebral artery. The position of the guide may be controlled by means of fluoroscopy or Polaroid films. The guide can be withdrawn and advanced again after changing the direction of the needle. If the resistance cannot be overcome, the guide should be left in position and the next step undertaken.

After the metallic guide is in place, it

should be held with one hand close to the distal part of the needle while the needle is withdrawn over it (Fig. 3*c*). Using slight rotatory movements, the previously prepared polyethylene tubing is inserted over the metallic guide, through the skin and arterial wall, and into the vascular lumen (Fig. 3*d*). The guide must be held firmly with the fingers of one hand while the catheter is advanced 4 or 5 cm. into the subclavian artery with the other. For injection of the innominate and right carotid arteries, the catheter should be advanced approximately 8 to 10 cm. If the aortic arch is to be visualized, the catheter should be advanced 13 to 15 cm. into the vessel. The catheter must not be advanced together with the metallic guide since tortuosity or kinking of the subclavian and innominate arteries will occasionally prevent the guide from easily following the course of the vessel. Also, since the catheter may enter the thyrocervical trunk or vertebral artery, advancing the guide and catheter together could injure the intima.

When the catheter is in the desired position, it is held with the fingers of one hand and the metallic guide withdrawn. Continued pulsatile blood flow, although less than that observed to flow through the needle, is a good indication that the catheter is in the correct position. Occasionally, after the metallic guide has been withdrawn, kinking of the catheter or its position in a smaller branching vessel will prevent back-flow. If this occurs, the catheter should be slowly pulled back 1 to 2 cm.

When assured that the catheter has been correctly placed, the one-way stopcock is fitted to the tubing adapter and connected to the 30 ml. syringe with the long plastic tube (Fig. 3*e*). When simultaneous bilateral injection is to be carried out, the catheters on both sides are connected to the syringe through the Y-shaped connecting tube (Fig. 3*f*). To prevent clotting in the tube, 1 or 2 ml. of normal saline solution containing heparin is injected at frequent intervals.

To be certain of the exact position of the catheter tip, it is advisable to make an ex-



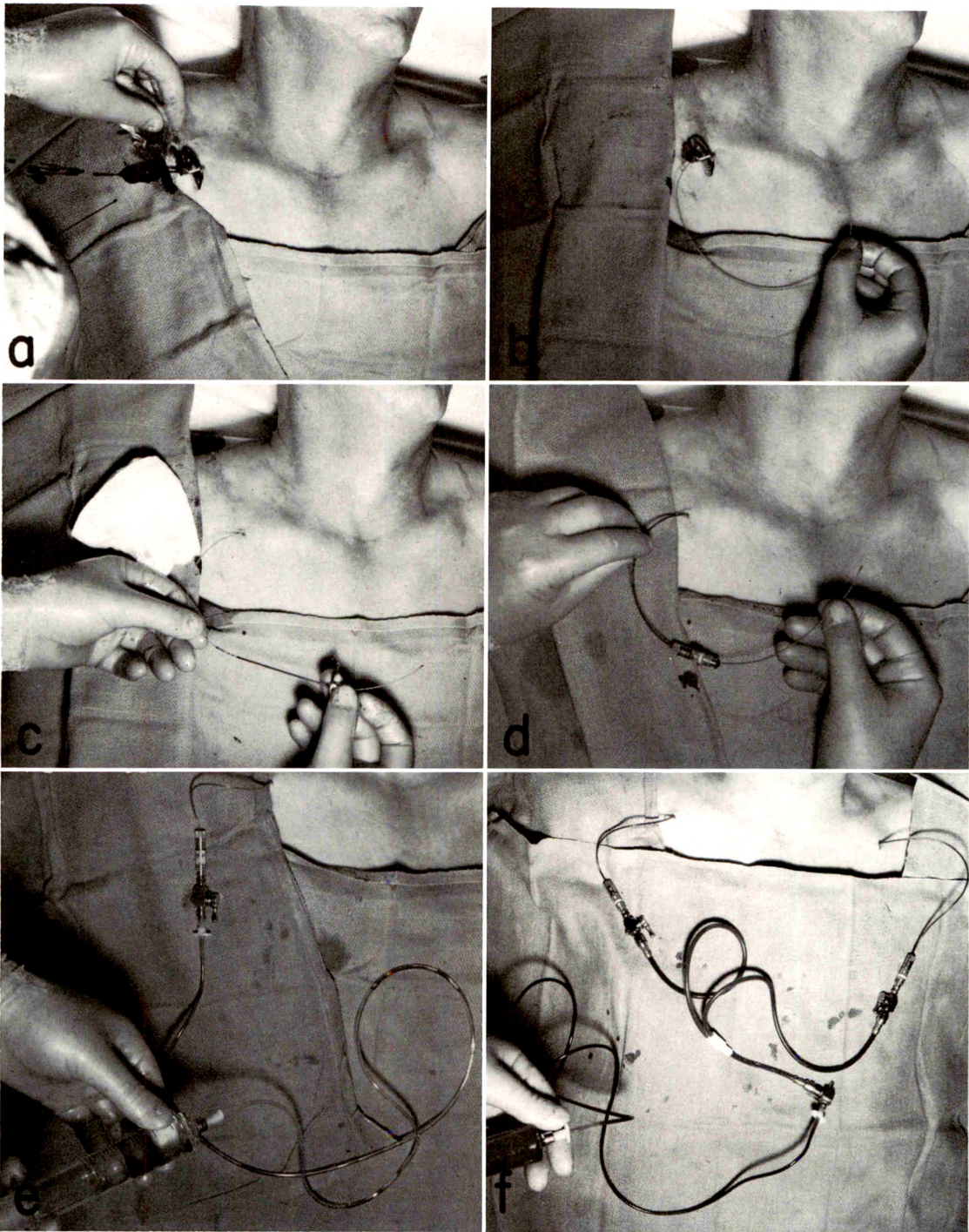


FIG. 3. Steps necessary for direct percutaneous infraclavicular catheterization of the subclavian artery. (a) Puncture of artery. (b) Insertion of metallic guide. (c) Withdrawal of needle. (d) Insertion of catheter. (e) Connection of polyethylene catheter to 30 ml. syringe for unilateral injection. (f) Connection of both catheters using a Y-shaped tube for bilateral simultaneous injection.



posure following an injection of a small amount of contrast material before proceeding with further injections. Twenty to 25 ml. of contrast material is used for unilateral injection and 30 ml. for bilateral simultaneous injection. The tubing system must be filled with contrast material before the amount desired for roentgenographic exposure is injected.

When both vertebral arteries are of large caliber, injection of one side may produce lamination of contrast material in the basilar artery (Fig. 9). In order to avoid this, a bilateral simultaneous subclavian injection is carried out (Fig. 4). Also, in patients in whom both vertebral arteries are hypoplastic or when the presence of stenotic lesions could prevent satisfactory visualization of the basilar artery and its branches, this procedure is preferred to unilateral injection (Fig. 5).

#### RESULTS

By employing this technique, we have been able to demonstrate several different types of lesions of the posterior cervicocranial circulation or posterior cranial fossa.



FIG. 4. Bilateral simultaneous injection of subclavian arteries to avoid lamination of contrast material in a case in which both vertebral arteries are large. A large fusiform aneurysm of the basilar artery is demonstrated.

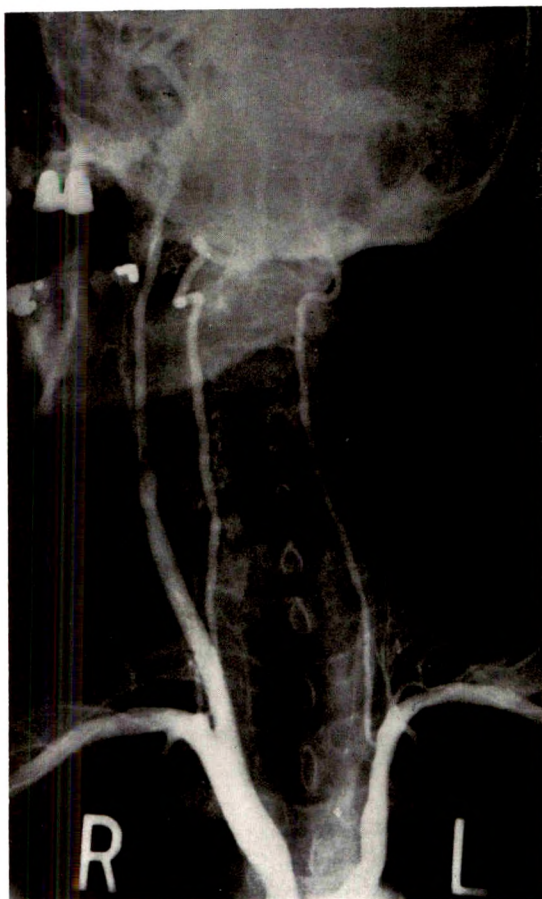


FIG. 5. Bilateral simultaneous injection of subclavian arteries revealing stenosis at origin of both small vertebral arteries.

1. *Atherosclerotic occlusive disease*, which may involve the vertebrobasilar system at different levels (Fig. 6, *a*, *b* and *c*). It frequently occurs in the proximal segment of the subclavian or innominate arteries near their origins or in the cervicocranial course of the vertebral arteries. It may also be present in the basilar artery or its branches. In cases of complete vertebral artery occlusion or when the vertebral arteries are very hypoplastic, the symptoms of vertebrobasilar insufficiency are often associated with occlusive disease of the carotid system, which prevents satisfactory collateral blood flow through the posterior communicating arteries. For this reason, when the symptoms are suggestive of vertebrobasilar insufficiency, the carotid arteries as well as



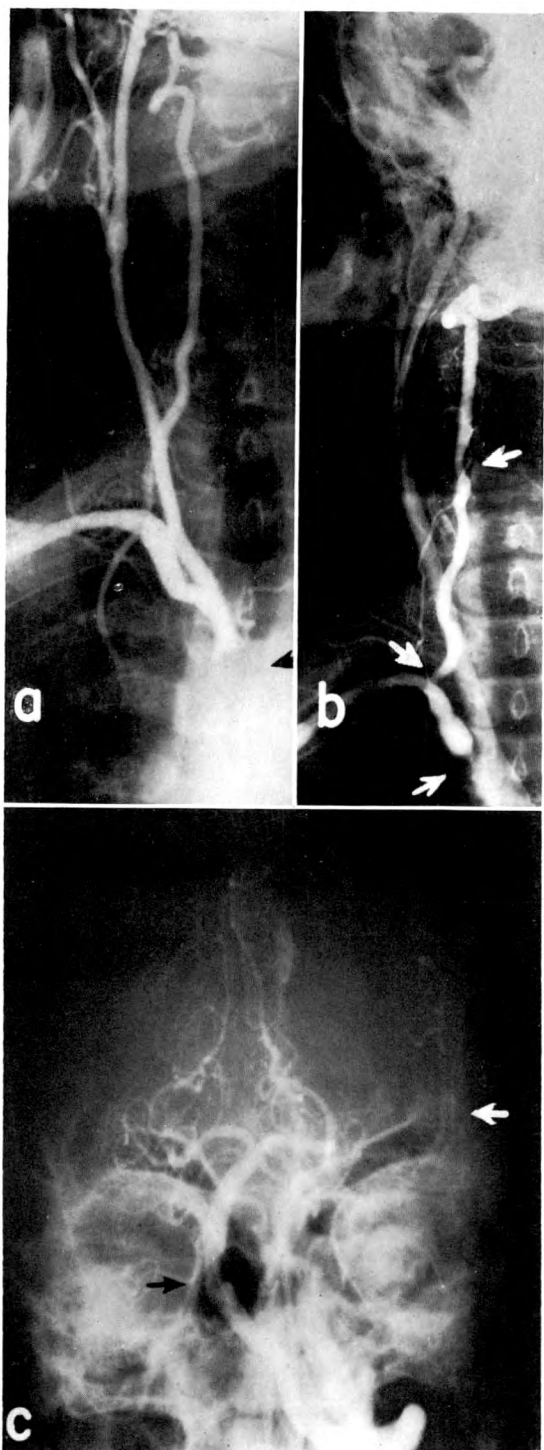


FIG. 6. Arteriograms in 3 patients showing occlusive disease at different levels. (a) Complete occlusion of the innominate artery (arrow). (b) Marked stenosis at origin of right subclavian artery (lower arrow), moderate stenosis at origin of right vertebral artery (mid arrow), and mild compression of the posterior circulation should be visualized.

the posterior circulation should be visualized.

2. Extrinsic compression by musculoskeletal structures occasionally produces *partial or complete occlusion* of the vertebral arteries. Patients with this type of lesion only complain of vertebrobasilar insufficiency when the head and neck are held in a certain position.<sup>1,2,4,5</sup>

Hyperextension of the head and neck, with or without rotation, may block the vertebral arteries in their proximal segment or at the base of the skull. This position may prevent visualization of the intracranial segment of the vertebrobasilar system in a submental-vertex view. Rotation of the head and neck to the side opposite that being injected may block the vertebral artery at the atlantoaxial joint on the side being injected (Fig. 7, *a* and *b*). On the other hand, rotation of the head to the side being injected may block the vertebral artery on that side in its proximal segment, preventing satisfactory visualization of the intracranial portion of the vertebrobasilar system, or it may give a false impression of complete occlusion of the vertebral artery at its origin.

For complete evaluation of patients with vertebrobasilar insufficiency symptoms associated with changes of position of the head and neck, cervicocranial angiography should be carried out with the head and neck first in hyperextension and then in a neutral position and rotated from one side to the other. These positions will assist in differentiating between mechanical compression and intrinsic stenosis or occlusion of the vertebral artery.

Cervical osteophytes occur most frequently at the C5-6 level. Atherosclerotic

sion of right vertebral artery by cervical osteophyte between C4-C5 (upper arrow). (c) Occlusive disease of the lower segment of the basilar artery (arrow on left) in a patient with a left vertebral artery only. There is collateral filling of the left middle cerebral artery (arrow on right) due to complete occlusion of the left internal carotid artery.

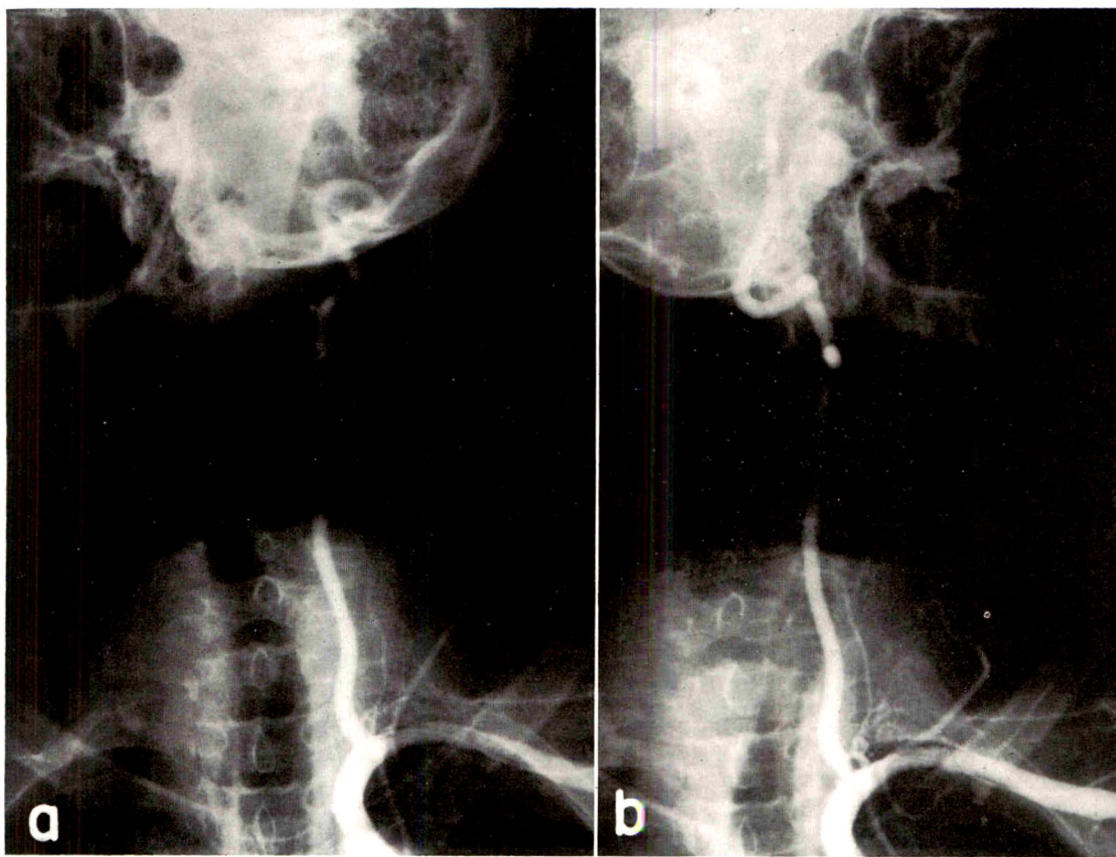


FIG. 7. Left subclavian artery injection showing (a) mechanical occlusion of the left vertebral artery at the level of atlantoaxial joint when the head and neck are rotated to the right side, and (b) satisfactory filling of the intracranial segment and basilar artery, after repeated injection, when the head and neck are rotated to the left side.

plaques may also occur at this level, and differential diagnosis is essential before surgical treatment can be considered. Extrinsic compression of the vertebral artery by cervical osteophytes occurs most often when the head and neck are turned to the side involved by the bony protrusion (Fig. 8, a and b). With the head in a neutral position or rotated to the side opposite the osteophyte, the alteration in caliber of the vessel is minimal or not observed at all. If narrowing at this level is produced by an atherosclerotic plaque, the caliber of the artery is unchanged and not affected by rotation of the head and neck in any direction. If angiography is carried out with the patient under general anesthesia, change in position of the head and neck must be passively maintained by a binder or tape.

This is usually unnecessary when the procedure is performed on a cooperative patient under local anesthesia.

3. *Aneurysm.* Fifteen intracranial saccular aneurysms of the posterior cervicocranial circulation were demonstrated. One was located on the fifth cervical radicular artery and one on the right anterior inferior cerebellar artery. Five were in the vertebral artery at the origin of the posterior inferior cerebellar artery, and 8 were at the bifurcation of the basilar artery (Fig. 9).

4. *Arteriovenous malformations* were demonstrated in 6 cases filling from the posterior circulation. In 4 the arterial blood supply was from the anterior as well as from the posterior part of the circle of Willis. Visualization of the feeding arteries in this



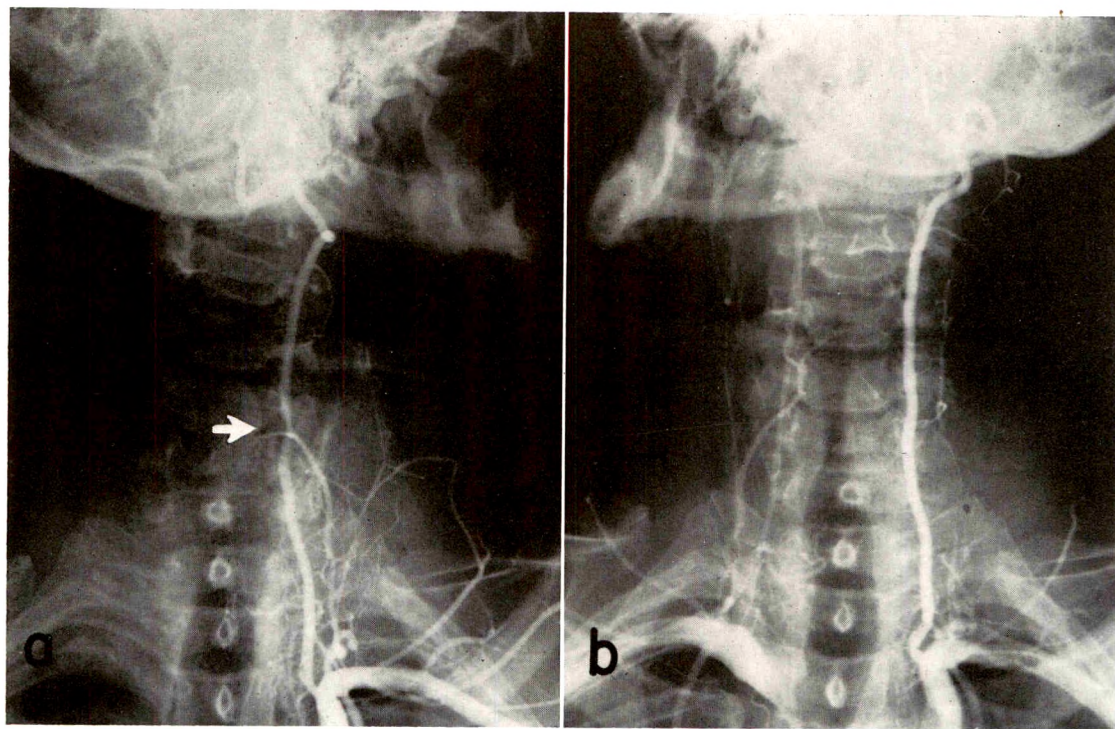


FIG. 8. (a) Injection of left subclavian artery demonstrating extrinsic compression of the left vertebral artery by a cervical osteophyte at level of C<sub>4</sub>-C<sub>5</sub> (arrow) when head and neck are turned to the left. (b) Repeat injection with the head to the right showing normal caliber of the artery throughout its course.

type of anomaly is particularly important if surgical treatment is contemplated (Fig. 10, a and b).

5. Angiography was of diagnostic value in 7 out of 9 cases with a prearteriographic

diagnosis of tumor of the posterior fossa. In 1 of the 2 remaining cases, the symptoms suggesting tumor were caused by a large arteriosclerotic fusiform aneurysm of the intracranial segment of the left vertebral artery (Fig. 11). No lesion was demonstrated in the remaining case.

#### COMPLICATIONS

In this series of 846 patients in whom a total of 1,631 catheterizations of the subclavian artery was performed using the infraclavicular approach, the complications were all local and minimal in the 74 patients (9.0 per cent) in whom they occurred. There were no deaths. Unilateral pneumo-

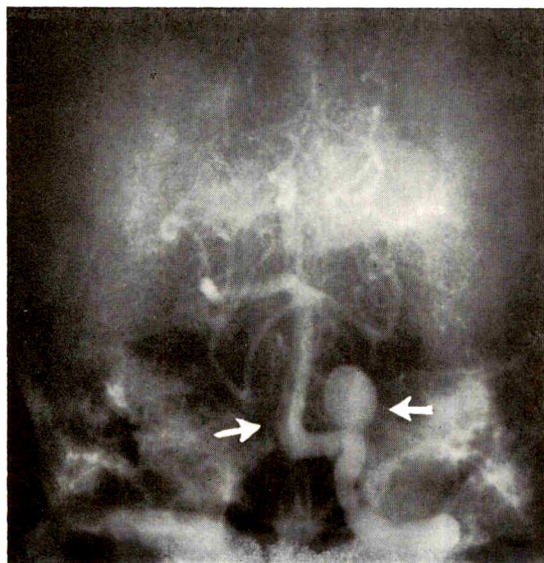


FIG. 9. Aneurysm of left vertebral artery adjacent to origin of posterior inferior cerebellar artery (arrow on right). Notice lamination of contrast material in the basilar artery (arrow on left).



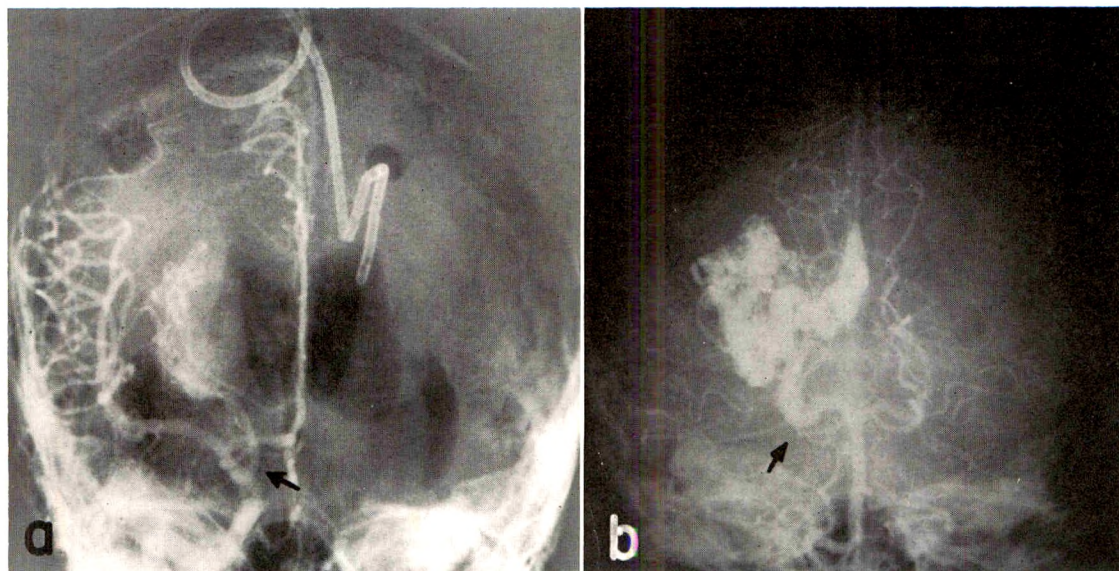


FIG. 10. (a) Arteriovenous malformation filling from a right carotid injection through a large anterior choroidal artery (arrow). (b) Right subclavian injection with filling through branches of right posterior cerebral artery (arrow).

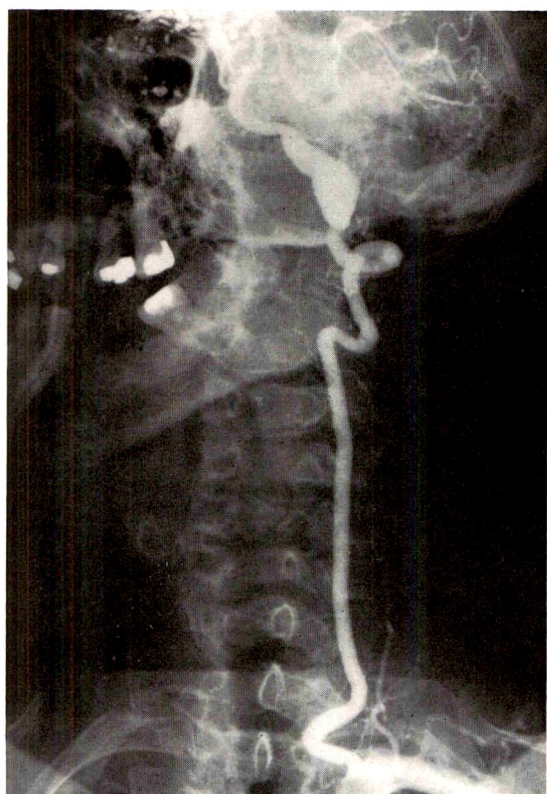


FIG. 11. Fusiform arteriosclerotic aneurysm (oblique view) of left vertebral artery at level of the foramen magnum. Prearteriographic diagnosis was posterior fossa tumor.

thorax occurred in 6 cases and bilateral pneumothorax in 1 case. A small unilateral hemothorax was disclosed in 1 patient 6 hours after arteriography. These complications occurred in 6 women and 1 man. The reason for the higher frequency in females is not clear. Small intramural injections or extravasation of the contrast material occurred in 54 cases. Hematoma after withdrawal of the catheter occurred in 12 cases, but this was readily controlled by manual compression of the artery. Cerebral complications, loss of brachial or radial pulse, arteriovenous fistula, or injury to the brachial plexus have not occurred in this series.

#### CONCLUSIONS

Direct percutaneous infraclavicular catheterization of the subclavian artery offers the following advantages:

1. It is easy to perform and essentially painless with local anesthesia if meglumine iothalamate 60 per cent (Conray) is the contrast medium used.
2. Expensive instruments are not required and routine roentgenographic equipment can be used.
3. This procedure permits positioning of



the head and neck in order to study the effect of musculoskeletal structures on the vertebral circulation or to obtain special projections without the risk of extravasation or injury to the arteries.

4. It is possible to perform bilateral simultaneous injection of the subclavian arteries in order to better visualize the basilar artery and its branches. Lamination of contrast material in the basilar artery can be avoided in cases in which both vertebral arteries are of the same caliber.

5. In cases in which only the right vertebral artery can be visualized due to anomalous origin, hypoplasia, or occlusion of the left vertebral, selective catheterization of the right vertebral can be carried out through the right subclavian artery to avoid having the contrast material enter the carotid system. This is critical when adequate delineation of the feeding arteries of an arteriovenous malformation is necessary, particularly when it receives its blood supply from the right carotid system as well as the posterior circulation.

6. This arteriographic technique has proved to be effective in visualizing aneurysms and arteriovenous malformations involving the posterior cranial circulation or in cases of suspected tumor of the posterior fossa. It permits localization of extrinsic compression of the vertebral artery by musculoskeletal structures.

7. The complications have been minimal and local.

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#### APPENDIX

##### EQUIPMENT REQUIRED FOR PERCUTANEOUS INFRACLAVICULAR CATHETERIZATION OF THE SUBCLAVIAN ARTERY

1. (a) Cournand-Grino needle, XT487-LNR, 17 gauge, 3½ inches long: Beckton-Dickinson and Co., Rutherford, New Jersey (Fig. 1a);
- (b) Seldinger needle, PE 160: Ohio

Chemical and Surgical Equipment Co., Division of Air Reduction, 1400 East Washington Avenue, Madison 10, Wisconsin (Fig. 1c).

2. Flexible metallic guide, 19 and 20 gauge, 80 cm. long, 8 to 10 cm. straight flexible tip: C. Christian & R. Tilbury, 2741 Kyle Avenue North, Minneapolis, Minnesota (Fig. 1, b and d).
3. (a) Polyethylene tubing, PE 190 or PE 200 caliber, 20 to 25 cm. long: Clay Adams, 141 East 25th Street, New York 10, New York (Fig. 1e).
- (b) Punching instrument, #17.889-½: Schick X-Ray Co., Inc., 444 North Lake Shore Drive, Chicago 11, Illinois (Fig. 1f).
4. (a) Tubing adapter, A-1025, size C: Clay Adams (see No. 3a) (Fig. 1g).
- (b) Removable fitting, #BR 80 or BR 90: United States Catheter and Instrument Co., Glenn Falls, New York (Fig. 1f).
- (c) Flexible plastic connecting tube, K-50 sterile extension tube #5420: Pharmaseal Laboratories, Glendale, California (Fig. 1j).
- (d) One-way stopcock, B.D. MS04-T: Beckton-Dickinson (see No. 1a) (Fig. 1l).
- (e) Adapter, B.D. 608/L: Beckton-Dickinson (Fig. 1m).
5. One-way stopcock, female Luer-Lok to male Luer-Lok, B.D. MS09: Beckton-Dickinson (Fig. 1h).
6. (a) Y-shaped connecting tube, Cly-Q-Pak, #4617: Abbott Laboratories, North Chicago, Illinois (Fig. 1n).
- (b) Y-shaped connection adapter, B.D. 439A, female Luer-lok to two male Luer-Loks: Beckton-Dickinson.
- (c) Two adapters, B.D. H/468L: Beckton-Dickinson (Fig. 1o).
- (d) Adapter, B.D. L/606: Beckton-Dickinson (Fig. 1p)

(e) 30 ml. syringe: Beckton-Dickinson (Fig. 19).

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## POST-ANGIOGRAPHY ISOTOPE BRAIN SCANNING\*

### POSITIVE OR NEGATIVE?

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**D**URING the past several years there has been general acceptance of radioisotopic brain scanning as a screening procedure in the evaluation of neurologic patients. Some of these patients have been subjected to cerebral arteriography prior to scanning. Recently, several reports have appeared in the medical literature concerning false positive isotopic brain scans due to artifacts related to previous cerebral arteriography.<sup>2,5,7</sup> These changes have been attributed to alterations in the blood brain barrier after the introduction of contrast material or to technical problems in the needle puncture technique. Because of confusion about whether this is a significant factor in routine brain scanning, 10 patients in whom pre- and post-arteriographic brain scans were made were evaluated. The authors report the results of this study.

#### METHOD

Brain scanning was performed in the usual manner using  $\text{Hg}^{197}$  in a dosage of 15  $\mu\text{c}/\text{kg}$ . as a part of the evaluation of patients with suspected cerebral lesions. Subsequently, cerebral arteriography was performed, and within the next few days, brain scanning was repeated. Patients were limited to those individuals who had negative pre-angiographic brain scans, and who were over 38 years of age. One younger patient (28 years of age) was rescanned for other reasons and is included in the series. Every attempt was made to avoid technical problems in needle puncture technique during arteriography. Most of the patients had preliminary roentgenograms of the neck to confirm optimal needle position. Renografin 60 was used in all patients.

The scans were interpreted independently of the patient's history and again after knowledge of the clinical findings.

#### DISCUSSION

It has been known for many years that intravascular contrast material can affect the blood brain barrier. Broman and Olsson<sup>3</sup> describe injections in dogs with increasing volumes of contrast material in which trypan blue given intravenously was shown to stain the brain, presumably related to alterations in the blood brain barrier. Two variables were indicated: the concentration of the contrast medium and the duration of time that the medium was in proximity to the brain. Other signs of brain toxicity have been used to gauge the effect of contrast material on the brain. Hilal<sup>4</sup> has used the production of seizures in dogs as an indication of excessive administration of intra-arterial contrast material.

Bender<sup>2</sup> states that following arteriography his scans were frequently uninterpretable. He stated that an entire hemisphere might have 20 to 30 per cent more activity than the opposite side, and that underlying lesions might be missed where the radioactivity was diffusely increased. McAfee<sup>6</sup> has also stated that ill-defined diffuse activity may be seen throughout the entire hemisphere following arteriography, or the study may be normal. He cautions that radioisotope scanning should not be performed until 3 to 7 days following arteriography, and states that he has never seen an abnormal scan if the abnormality was due to an earlier arteriography when this precaution was followed.

There is significant evidence<sup>1</sup> to suggest that faulty needle technique may be the most significant factor in complications relating to carotid arteriography. This usually is caused by subintimal dissection which can partially or wholly obstruct the arterial lumen. Monitoring of each puncture by a lateral roentgenogram, particu-

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TABLE I

Patient	Age (yr.)	Disease	Vessels Punctured	Volume of Contrast Material (cc.)	Neurologic Sequelae	Interval following Arteriography	Post-angiography Scan
F.H.	40	Left focal seizures	RCC	20	None	18 da.	neg.
M.B.	43	Nasopharyngeal cancer	LCC	20	None	11 da.	neg.
J.G.	73	Headache	RCC LCC	20 20	None	2 da.	neg.
F.C.	38	Left hemiparesis; right headache	RCC	20	None	2 da.	neg.
A.R.	63	Headaches	RCC LCC Vertebral	20 20 16	None	2 da.	neg.
L.C.	51	Occlusive disease	RCC LCC	20 20	None	2 da.	neg.
D.J.	52	Glioma	RCC	20	None	2 da.	neg.
T.S.	41	Seizures	RCC LCC	20 20	None	2 da.	neg.
J.D.	56	Temporal lobe seizures; coronary disease	RCC LCC	20 20	None	2 da.	neg.
W.M.	28	Aneurysm	RCC LCC Right brachial	20 20 40, 40	None	14 da.	neg.

LCC=left common carotid.  
RCC=right common carotid.

larly if the film can be rapidly developed, will allow re-position of the needle, with immediate correction of the hemodynamic alteration. Failure to observe these artifacts may be responsible for many of the complications related to carotid arteriography, and, until recently, attributed to contrast material. With partial occlusion of the common carotid artery, one could readily envision transient ischemia of the entire hemisphere leading to changes in the blood brain barrier. The scan would reflect these changes. Fortunately, many of these arterial artifacts are quickly reversible over the next several days. The clinical alterations may be minimal and are often dismissed.

McAfee and Taxdal<sup>5</sup> have also noted changes in the brain scan made several days after pneumoencephalography.

In brief, this study, which involves a very limited number of patients, suggests that routine, carefully performed, carotid arteriography does not produce abnormalities in the post-angiography isotopic brain scan. It is suggested that when diffuse areas of radioactivity are seen following angiography, it may be a reflection of transient arterial compromise related to the puncture technique, as well as the more widely publicized toxicity due to excessive contrast material. Changes in the blood brain barrier may occur in the area supplied by the carotid artery and these areas may



show increased radioactivity on the brain scan.

#### SUMMARY

In the last several years there have been statements in the medical literature that brain scans may demonstrate variable increases in radioactivity following carotid arteriography. It is possible that some of these areas of increased radioactivity might be interpreted as "false positives."

Ten patients are reported in whom routine neurologic evaluation by scanning showed no evidence of abnormal radioactivity and cerebral arteriography followed by repeat brain scans with  $\text{Hg}^{197}$  likewise showed no evidence of abnormal radioactivity. In those patients in whom greater quantities of contrast material are given or in whom a technical artifact is created by a faulty arterial puncture, certainly, it is possible that cerebral injury with alterations of the blood brain barrier might be produced. However, in well performed, routine, cerebral arteriography, this is probably not a significant factor.

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## BRAIN ABSCESS IN CHILDREN\*

### RADIOLOGIC METHODS FOR EARLY RECOGNITION

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**R**ECENTLY, we have had the opportunity to study 4 patients with the diagnosis of cyanotic congenital heart disease who had symptoms of intracranial involvement by a process which was suspected of being a brain abscess.

To our knowledge, previous reports have not specifically noted the use of the radioisotope brain scans in this particular clinical situation. It is our wish to document our experience with radioisotope brain scanning as an earlier diagnostic procedure in this group of patients than arteriography and air encephalography. Reference will be made to the time interval between these examinations.

#### METHOD

On the evening prior to examination, 0.5 to 1.0 ml. of intramuscular mercurhydrin is administered. On the following morning, 10  $\mu$ c/kg. body weight of  $\text{Hg}^{197}$  chlormerodrin is injected intravenously. Scanning begins within 3 to 4 hours after this injection. We first obtain an anterior or posterior scan, and if lateralization of activity is noted, then a lateral scan of that side is made. If no lateralization occurs, then scans are made in the anterior, posterior, right and left lateral positions. A Picker Magna Scanner equipped with a 3×2 inch sodium iodide thallium activated crystal is used. This is equipped with a lead shielded 19 hole collimator and a pulse height analyzer. Other details of scanning and reference to radiation dose have previously been described.<sup>20</sup>

#### REPORT OF CASES

**CASE I.** L.D., a 7½ year old white female, was seen first at The Children's Hospital Medical Center of Boston at the age of 7 months with

symptoms of easy fatigability, rapid breathing and cyanosis. A diagnosis of hemitruncus arteriosus was made with the aid of cardiac catheterization.

She was seen frequently thereafter and was clinically well until 5 days prior to her most recent admission, when she developed generalized headaches, most marked over the left hemisphere and a temperature of 103°F. Delirium, "random speech" and some diminution in visual acuity were associated symptoms.

On admission, her vital signs were: blood pressure 100/60, respiration 24/min., pulse 88/min., and temperature 99.6°F. Positive physical findings were: 2+ cyanosis with clubbing of the extremities and a Grade III continuous murmur in the fourth right intercostal space which was transmitted to the right chest. Except for right hemianopsia, the neurologic examination was otherwise not remarkable.

Laboratory examination revealed a white blood cell count of 10,200 with 62 per cent polymorphonuclear cells, 1 per cent bands, 35 per cent lymphocytes and 2 per cent monocytes. The hematocrit was 58 per cent.

Lumbar puncture revealed an opening pressure of 110 mm. of water and a closing pressure of 105 mm. There were 24 white blood cells per high power field, 18 monocytes and 6 polymorphonuclears. The total protein of the spinal fluid was 71 mg. per cent and the sugar was 50 mg. per cent.

On the second hospital day, a radioisotope brain scan disclosed selective uptake of radioactivity in the left posterior parietal lobe (Fig. 1, *A* and *B*). Electroencephalography, performed on the same day, revealed "reversal of high voltage delta waves in the left posterior temporal region." A sonar encephalogram showed a 3 mm. shift of the midline from the left to the right.

She began treatment with intravenous penicillin (6 million units twice daily) and chloramphenicol (400 mg. twice daily). Dilantin, 25 mg. twice daily, was also administered.

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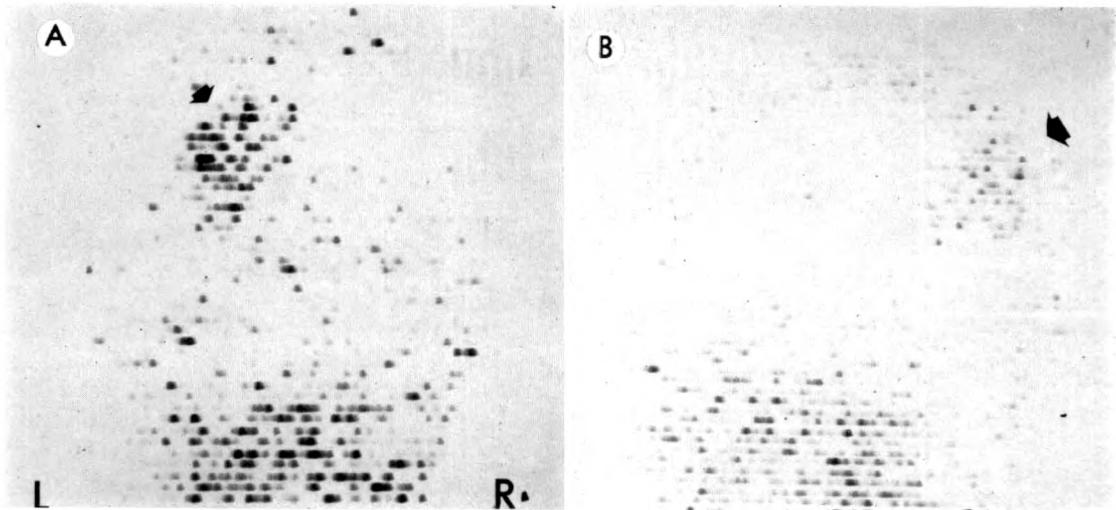


FIG. 1. Case I. (A) Posterior scan made on the second hospital day. There is an area of activity to the left of the midline (arrow). (B) Left lateral scan. The localized activity is in the posterior parietal lobe (arrow).

Over the next 4 days, her clinical condition remained stable. At this time, through a left parietal-occipital burr hole, a brain cannula was passed into the area of localized activity demonstrated on the isotope scan. No mass or purulent material was encountered on three attempts. A tentative diagnosis of focal cerebritis was made and she was continued on intravenous antibiotics. On the tenth hospital day, a repeat brain scan revealed a more diffuse pattern

of localization of radioisotope (Fig. 2, A and B). It was speculated that this widened area of increased isotopic uptake might be due either to extension of the cerebritis or to contusion and hemorrhage from the attempted aspirations. An electroencephalogram at this time also seemed to reveal a more diffuse focus.

On the 16th hospital day, as her condition remained stable, oral antibiotics (400,000 units of penicillin twice daily and 200 mg. of chloromy-

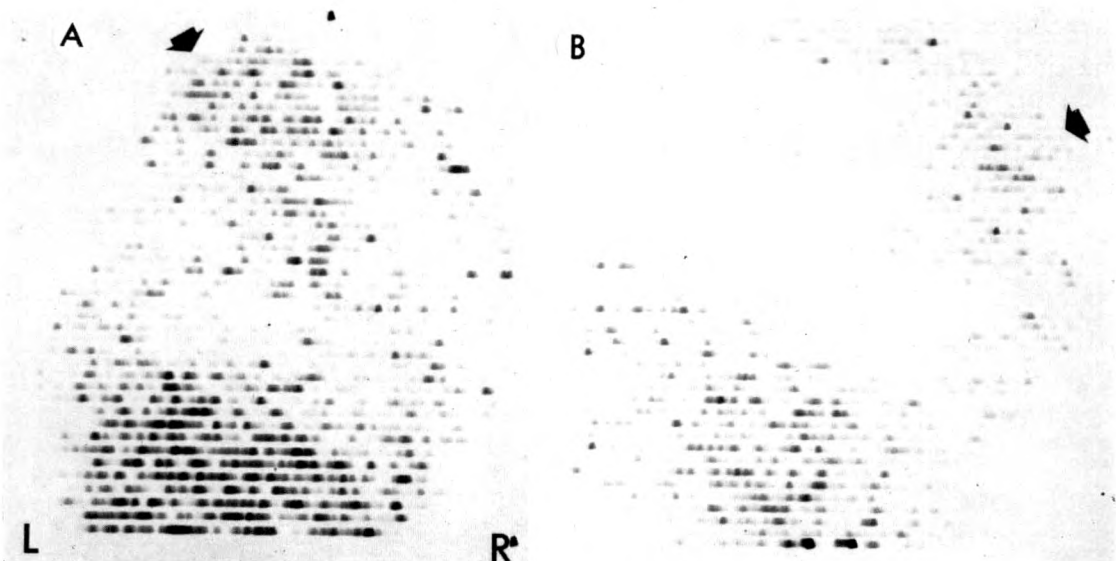


FIG. 2. Case I. (A) Posterior scan made on the 10th hospital day. The area of activity, lateralized to the left hemisphere, appears more diffuse than on previous examination (arrow). (B) Left lateral scan. The same increased area of uptake again is noted in the left posterior parietal lobe (arrow).

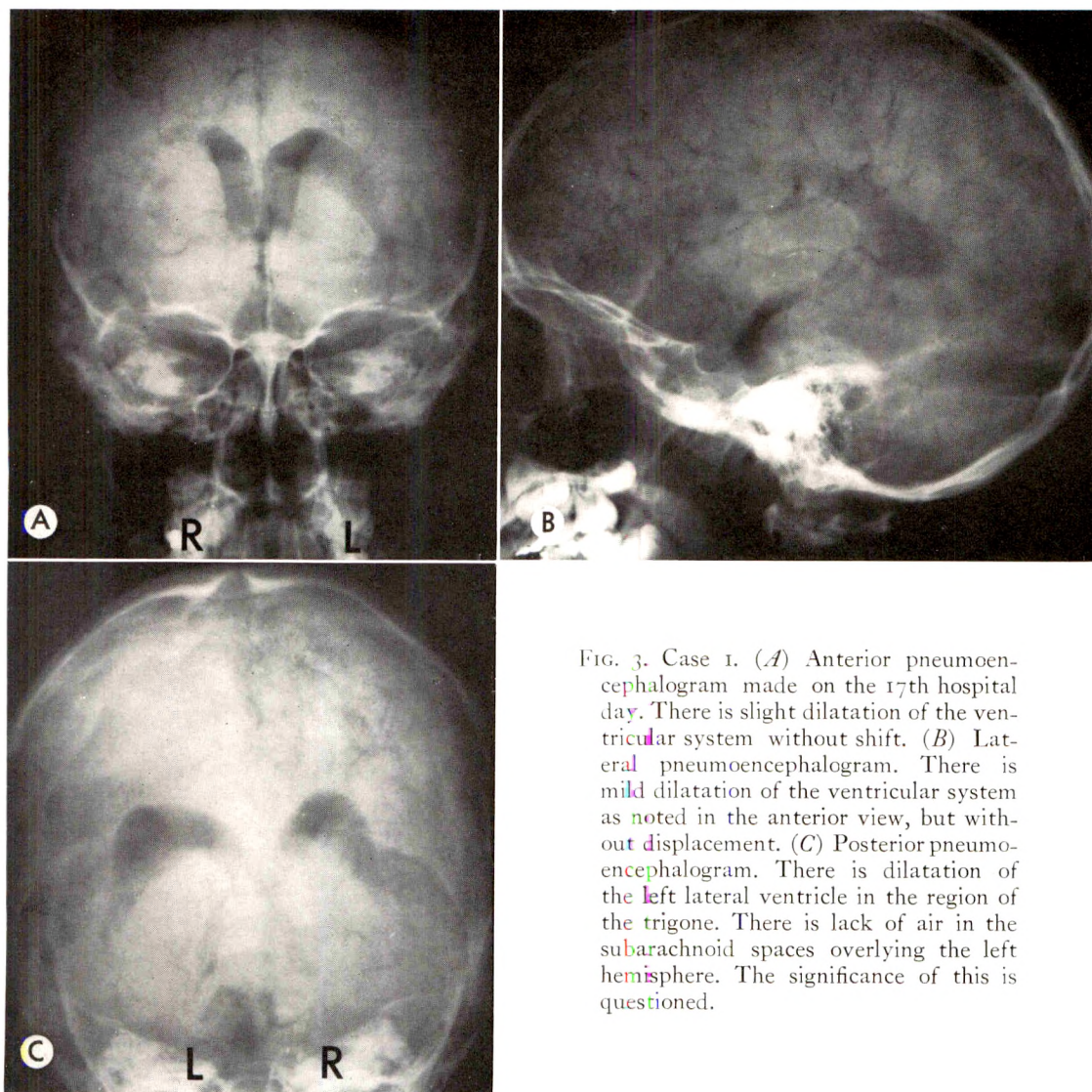


FIG. 3. Case 1. (A) Anterior pneumoencephalogram made on the 17th hospital day. There is slight dilatation of the ventricular system without shift. (B) Lateral pneumoencephalogram. There is mild dilatation of the ventricular system as noted in the anterior view, but without displacement. (C) Posterior pneumoencephalogram. There is dilatation of the left lateral ventricle in the region of the trigone. There is lack of air in the subarachnoid spaces overlying the left hemisphere. The significance of this is questioned.

cetin twice daily) were substituted for intravenous medication.

On the 17th hospital day, the electroencephalogram revealed a more localized abnormal tracing and the sonar encephalogram showed less shift of midline structures. At this same time, a pneumoencephalogram revealed minimum dilatation of the left lateral ventricle in the region of the trigone. No definite shift of the midline structures or displacement of the ventricular system was seen. There was noted to be lack of air in the subarachnoid spaces over the left hemisphere but this finding was considered of doubtful significance. The dilatation of the left ventricle was considered to be due possibly

to cerebral atrophy secondary to a long standing process (Fig. 3, A, B and C).

On the 23rd hospital day, it was decided to discontinue antibiotics for a trial period to determine whether discrete localization of infection would become evident and allow more definitive surgical treatment.

She remained well until the 33rd hospital day when her temperature rose to 101.8°F, while her peripheral white blood cell count and differential remained essentially unchanged. The electroencephalogram now revealed an increased focus of abnormality and the sonar encephalogram showed a 3.5 mm. shift of midline structures to the right. After antibiotics



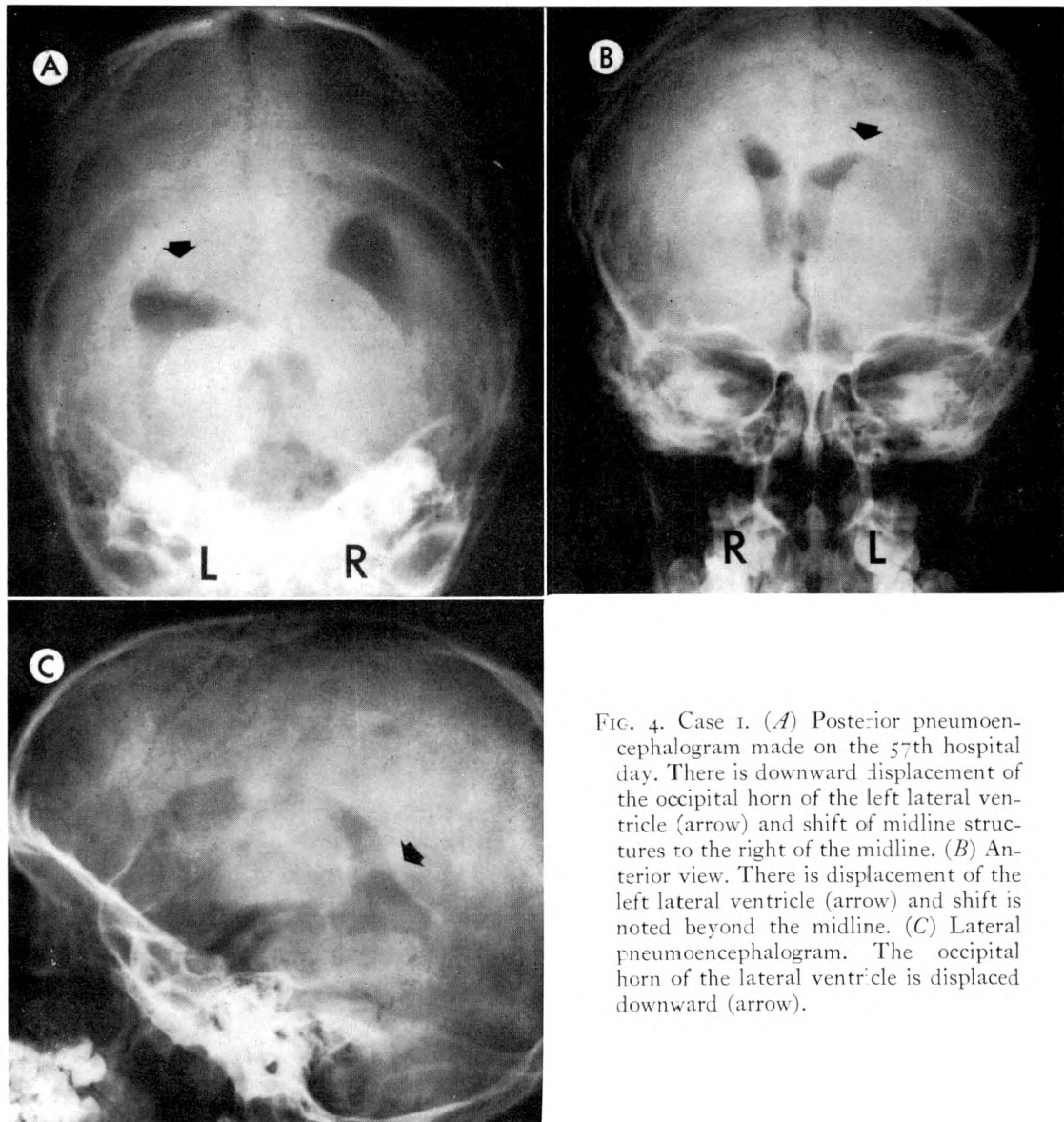


FIG. 4. Case 1. (A) Posterior pneumoencephalogram made on the 57th hospital day. There is downward displacement of the occipital horn of the left lateral ventricle (arrow) and shift of midline structures to the right of the midline. (B) Anterior view. There is displacement of the left lateral ventricle (arrow) and shift is noted beyond the midline. (C) Lateral pneumoencephalogram. The occipital horn of the lateral ventricle is displaced downward (arrow).

were reinstituted, her fever gradually fell towards normal. A repeat brain scan on the 47th hospital day showed uptake as before.

A repeat pneumoencephalogram on the 57th hospital day now revealed downward displacement of the left occipital horn with a shift of midline structures to the right (Fig. 4, A, B and C). This was thought to indicate that there was now a sizable mass lesion in the area of increased isotope uptake. At this time, repeat aspiration produced 40 ml. of purulent material from the area of previous negative tap. This contained Gram negative cocci on smear and cultured

*Streptococcus faecalis*. One cc. of thorotrast was instilled into the cavity in order to outline it and follow healing<sup>6,8,13</sup> (Fig. 5, A, B and C). Subsequent roentgenograms showed some re-accumulation and an additional 20 ml. of purulent material was aspirated. The lesion continued to diminish to about 1 cm. in diameter and it was decided that surgical excision was not indicated. She had an uneventful hospital course and was discharged on the 88th hospital day on maintenance antibiotics. At this time, her only positive neurologic sign, that of right lower homonymous quadrantanopsia, had diminished.

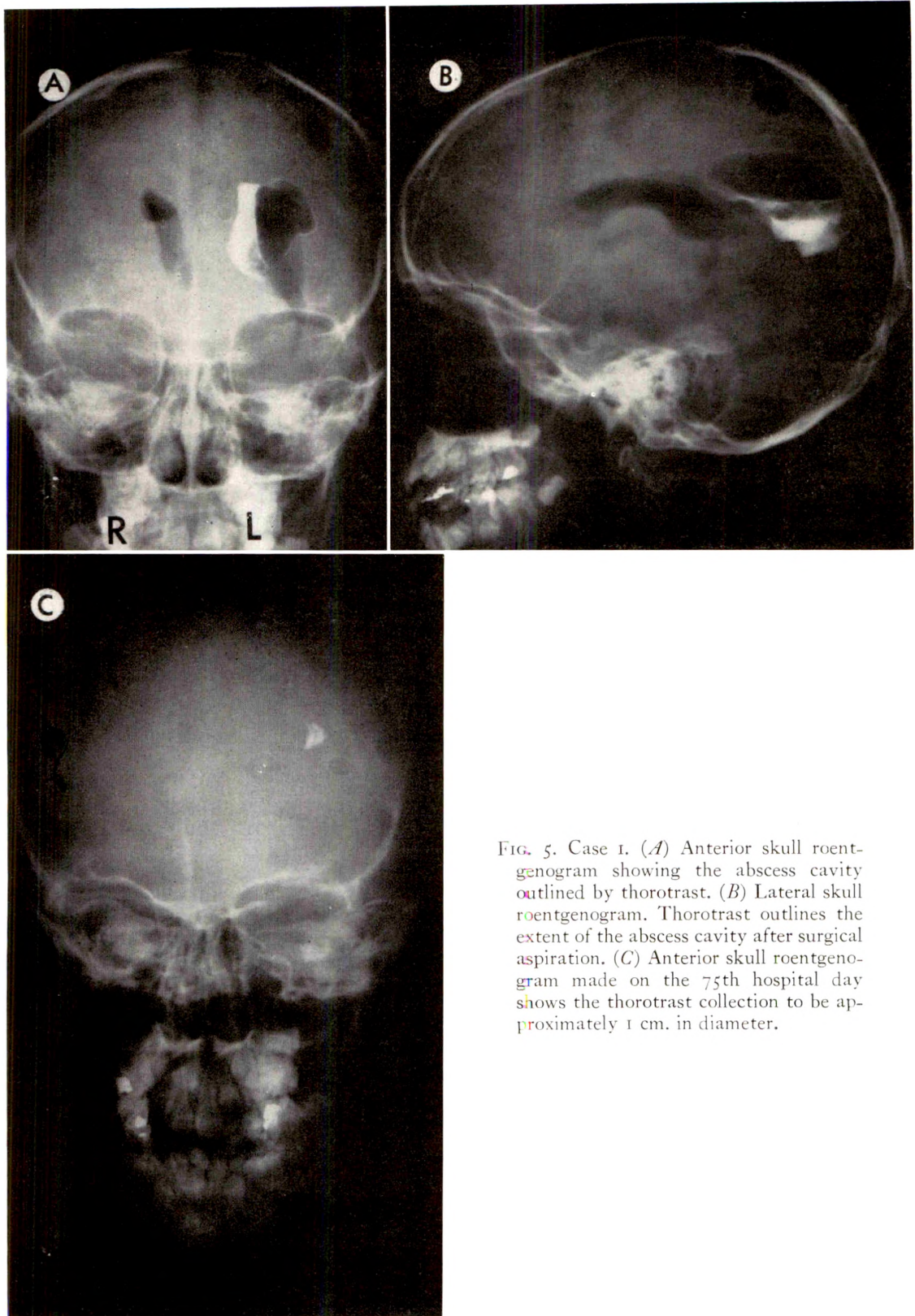


FIG. 5. Case 1. (A) Anterior skull roentgenogram showing the abscess cavity outlined by thorotrast. (B) Lateral skull roentgenogram. Thorotrast outlines the extent of the abscess cavity after surgical aspiration. (C) Anterior skull roentgenogram made on the 75th hospital day shows the thorotrast collection to be approximately 1 cm. in diameter.



CASE II. S.B., a white female child, was first seen at The Children's Hospital Medical Center when she was 8 days of age because of cyanosis from the age of 2 days. Following evaluation, which included cardiac catheterization, a diagnosis of transposition of the great vessels, with a small atrial shunt, was made. Therapy was directed to the creation of an atrial septal defect in order to increase the shunt.

Following discharge, she was maintained on digoxin. Careful follow-up evaluation showed no signs of cardiac decompensation.

Two weeks prior to her present admission when she was 4 years of age, she began to complain of intermittent frontal headaches and was said to have a "fever." Vomiting and diminished activity were noted 4 days prior to admission.

On admission, the vital signs were: blood pressure 112/68, pulse 88/min., respiration 30/min., and temperature 99.4°F. Positive findings on physical examination were: cyanosis and clubbing of the extremities; a 2+ right ventricular impulse; a thrill to palpation at the left sternal border; S1 increased 2+ and maximum at the left sternal border; a Grade II systolic murmur at the left sternal border; and a Grade III diastolic murmur at the left lower sternal border which was transmitted to the upper left sternal border and the apex of the left lung. Neurologic examination was not remarkable.

Laboratory findings were: white blood cell count of 9,050 with a differential of 83 per cent polymorphonuclears, 10 per cent lymphocytes, 4 per cent monocytes and 3 per cent bands. The hematocrit was 74 per cent.

Lumbar puncture revealed an opening pressure of 250 mm. of water and a closing pressure of 190 mm. The Pandy test was 2+. Total protein of the spinal fluid was 40 mg. per cent and the sugar was 53 mg. per cent. There were 330 white blood cells per high power field with a differential of 250 polymorphonuclears and 80 monocytes. Smear and culture of the spinal fluid were negative.

The electroencephalogram was "consistent with increased intracranial pressure." In addition, it showed a slow wave focus over the right mid-temporal area "consistent with a mass in this area."

An Hg<sup>197</sup> brain scan revealed selective uptake in the right temporal lobe (Fig. 6, A and B).

Penicillin (2 million units intravenously every 2 hours) and chloramphenicol (250 mg. intravenously every 6 hours) were prescribed. On the third hospital day, she developed a protracted left-sided paralysis following a left-sided seizure.

On the fourth hospital day, a brain cannula was introduced into the right temporal lobe through a burr hole and 10 ml. of purulent material was aspirated from what appeared to be a well encapsulated lesion, approximately 1.5 cm. deep to the dura. This cultured *Hemophilus parainfluenzae*.

While on the operating table, she developed cardiac arrest and died. At autopsy, approximately 8 separate abscesses were seen to be scattered throughout the brain substance. Some of these were as large in diameter as the one depicted by the brain scan.

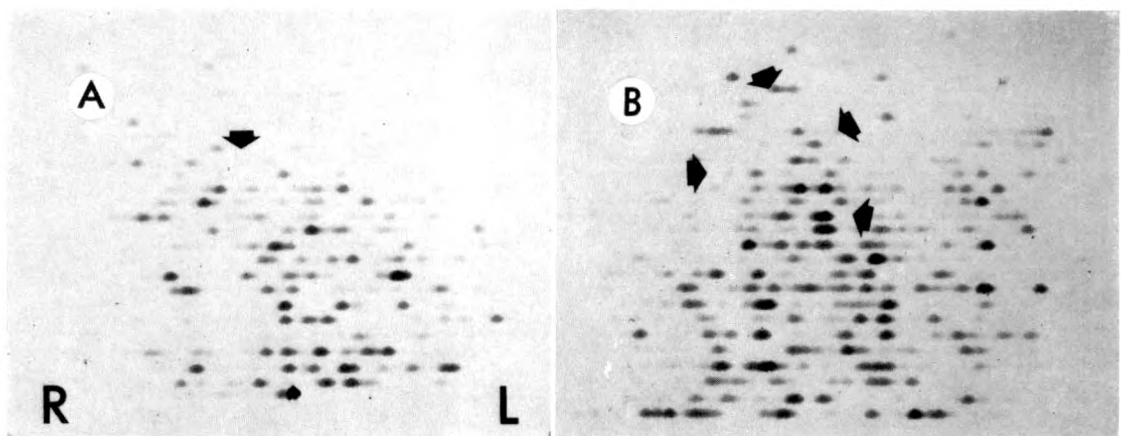


FIG. 6. Case II. (A) Anterior scan. There is localized uptake in the right hemisphere (arrow). (B) Right lateral scan. The selective uptake of the isotope is located in the temporal lobe (arrows).

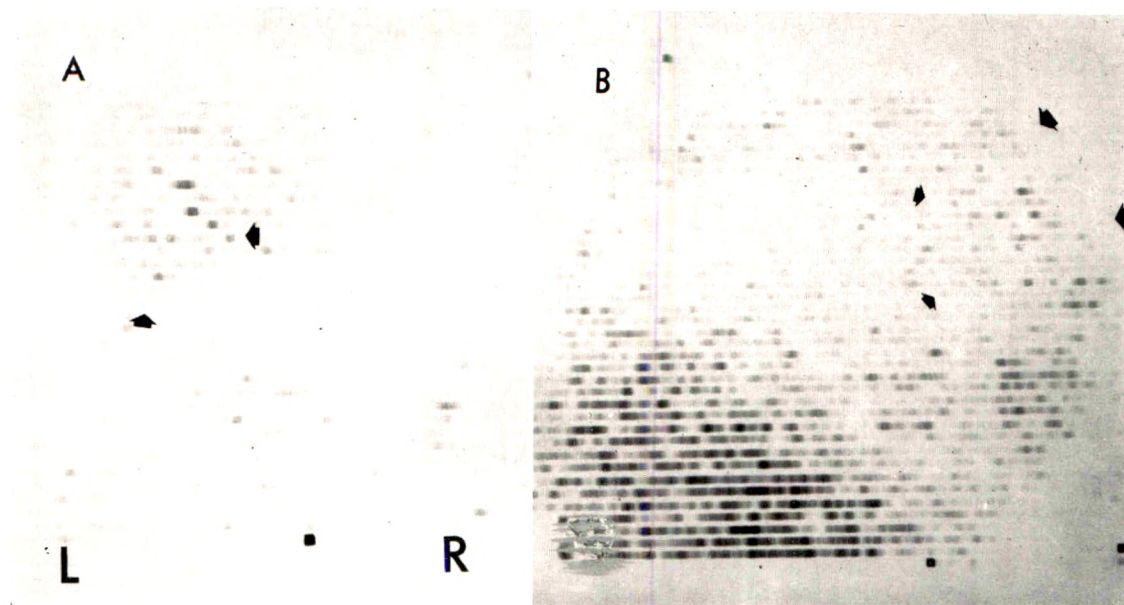


FIG. 7. Case III. (A) Posterior scan made shortly after admission shows selective uptake of isotope lateralized to the left hemisphere (arrows). (B) Left lateral scan. The selective uptake is localized to the posterior parietal lobe (arrows).

CASE III. G.S., a Negro male was first seen at The Children's Hospital Medical Center at the age of  $2\frac{1}{2}$  years, with a history of premature birth, cyanosis, clubbing of the extremities and poor growth.

Evaluation at that time, which included cardiac catheterization, revealed transposition of the great vessels with pulmonic stenosis and a ventricular septal defect.

Following discharge, he did well until 6 days prior to his current admission when at the age of  $3\frac{1}{2}$  years, he developed a right-sided motor seizure and "fever."

On this admission, physical examination revealed vital signs of: blood pressure 110/30, pulse 80/min., respiration 20/min., and temperature  $100^{\circ}\text{F}$ . Positive findings were limited to the cardiac system: there was a systolic precordial thrill; the point of maximum impulse to palpation was in the mid-clavicular line; a Grade III-IV continuous murmur was noted in the aortic area; a Grade III-IV pansystolic pulmonic murmur was heard.

The electroencephalogram revealed a left posterior delta focus. The sonar encephalogram showed a 1 mm. shift of midline structures to the right.

An  $\text{Hg}^{197}$  chlormerodrin brain scan revealed localized uptake in the left posterior parietal lobe (Fig. 7, A and B).

Antibiotic therapy with penicillin (400,000 units every 6 hours) and chloramphenicol and dilantin, 3 times daily, was undertaken.

On the 7th hospital day, a pneumoencephalogram showed mild dilatation of the left lateral ventricle with possibly some distortion in the region of the roof of the trigone, giving a double contour appearance. No definite shift of structures beyond the midline was noted (Fig. 8). Spinal fluid examination, at this time, revealed a protein content of 13.8 mg. per cent and a sugar of 55 mg. per cent. There was one lymphocyte per high power field. Dynamics were within normal limits.

Antibiotic therapy was continued until the 16th hospital day, during which time his clinical condition was stable. On this day, all antibiotics were discontinued in an attempt to allow the process to become amenable to surgical management.

On the 20th hospital day, he developed a right sided clonic seizure which involved the face, arm and leg. After this, he had a definite hemiparesis and hemianopsia. A repeat brain scan at this time again showed localized activity in the left parietal lobe posteriorly, perhaps somewhat better localized than on the previous examination (Fig. 9, A and B).

A spinal puncture on the 21th hospital day revealed a protein content of 50 mg. per cent



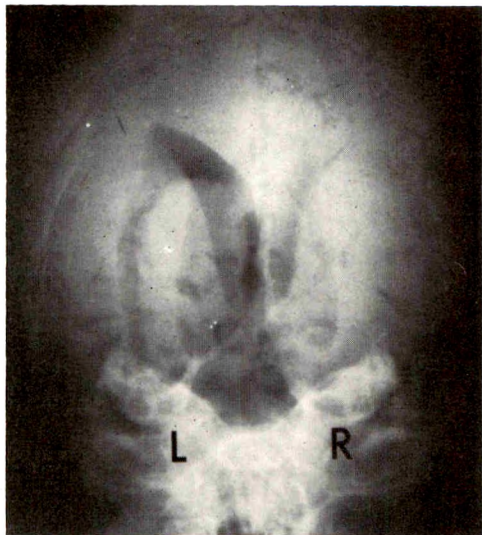


FIG. 8. Case III. Posterior pneumoencephalogram. Examination on the 7th hospital day shows dilatation of the left ventricular system without definite shift of midline structures.

and a sugar content of 47 mg. per cent. There were 60 polymorphonuclear cells per high power field. Repeat air study now disclosed a definite shift of midline structures to the right of the midline. There appeared to be a mass lesion which extended into the left lateral ventricle from the posterior parietal lobe (Fig. 10).

Exploration was undertaken the following day; 20 ml. of purulent material was aspirated through a burr hole placed over the left posterior parietal lobe. Biopsy revealed glial tissue with some perivascular inflammatory reaction. One cc. of thorotrast was instilled into the cavity (Fig. 11, A and B).

During his postoperative course, 2 additional aspirations of the abscess cavity were performed with removal each time of approximately 4 ml. of purulent material. Otherwise, his clinical course was unremarkable and by the 29th hospital day his right homonymous hemianopsia had cleared. He has remained asymptomatic subsequently and at 2 months, the thorotrast

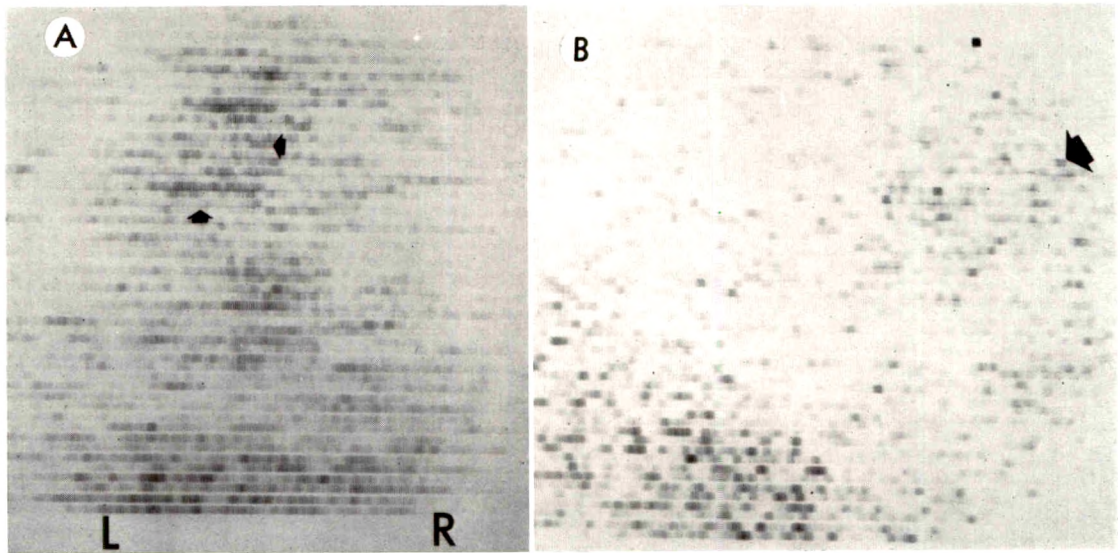


FIG. 9. Case III. (A) Posterior scan made on the 20th hospital day. Once again, there is selective uptake lateralized to the left of the midline arrows. (B) Left lateral scan. The area localized in the posterior parietal lobe is somewhat better demarcated than on previous examination 13 days ago (arrow).



FIG. 12. Case IV. (A) Anterior scan. There is selective localization of radioisotope to the right of the midline and superficially located in the cortex (arrow). (B) Right lateral scan. There is isotopic localization in the area of the posterior frontal lobe (arrow).



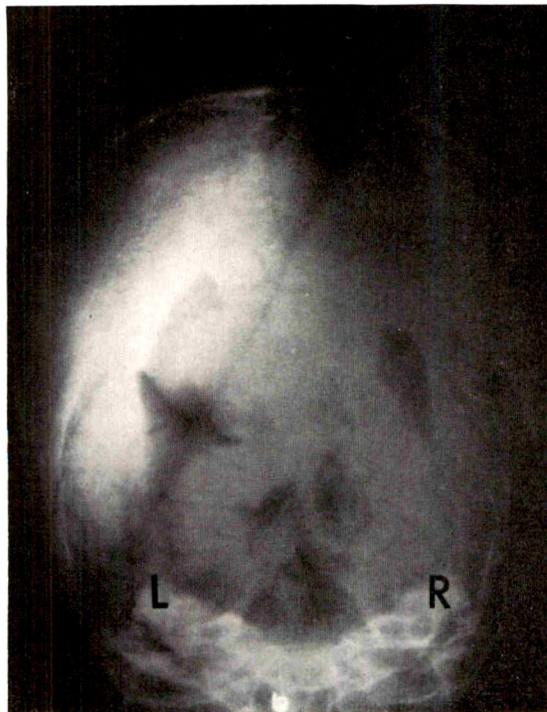
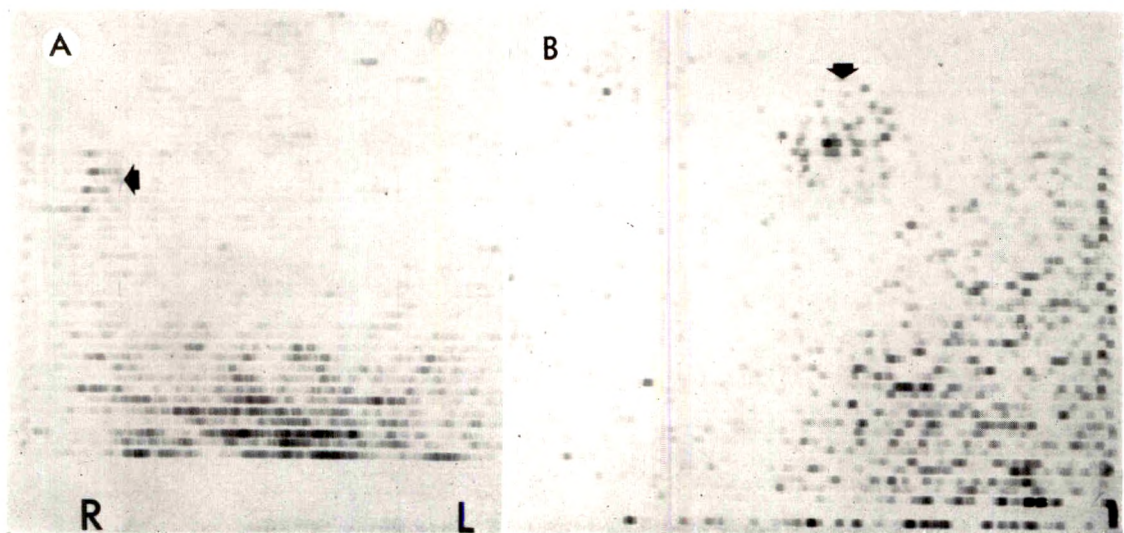
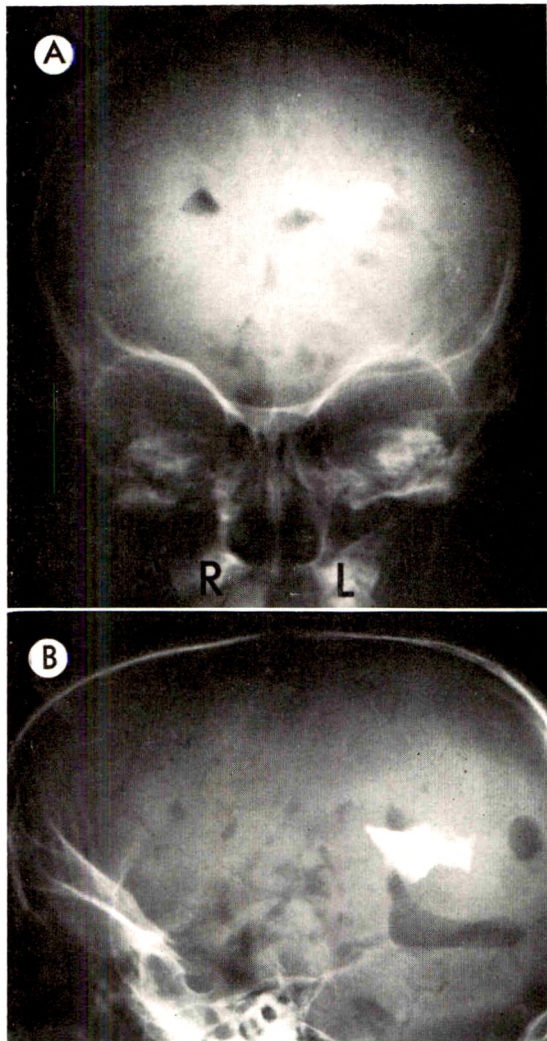


FIG. 10. Case III. Posterior pneumoencephalogram. Study performed 14 days after the original shows definite shift of midline structures to the right by a mass which extends into the left lateral ventricle, between it and the temporal horn.

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FIG. 11. Case III. (A) Anteroposterior skull roentgenogram shows thorotrast outlining the abscess cavity after surgery. (B) Lateral skull roentgenogram. The extent of the abscess cavity is noted in the lateral view by contrast with thorotrast.





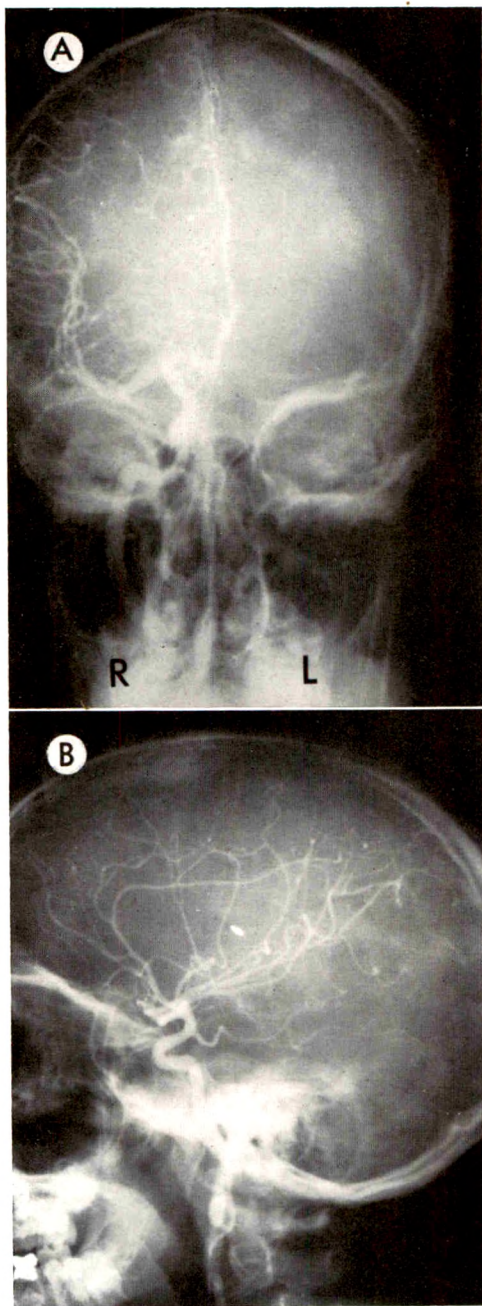


FIG. 13. Case IV. (A) Posterior and (B) lateral carotid arteriograms. There is slight displacement of the anterior cerebral artery to the left of the midline. In addition, the sylvian vessels may be distorted by a possible lesion in the right frontal lobe.

stained tissue was a lesion that measured approximately 1 cm. in diameter, deep in the left parietal lobe. It was felt that surgical excision was not indicated.

CASE IV. R.C., a white male, was first seen at The Children's Hospital Medical Center when he was 6 years of age because of cyanosis and "squatting." At that time, a diagnosis of

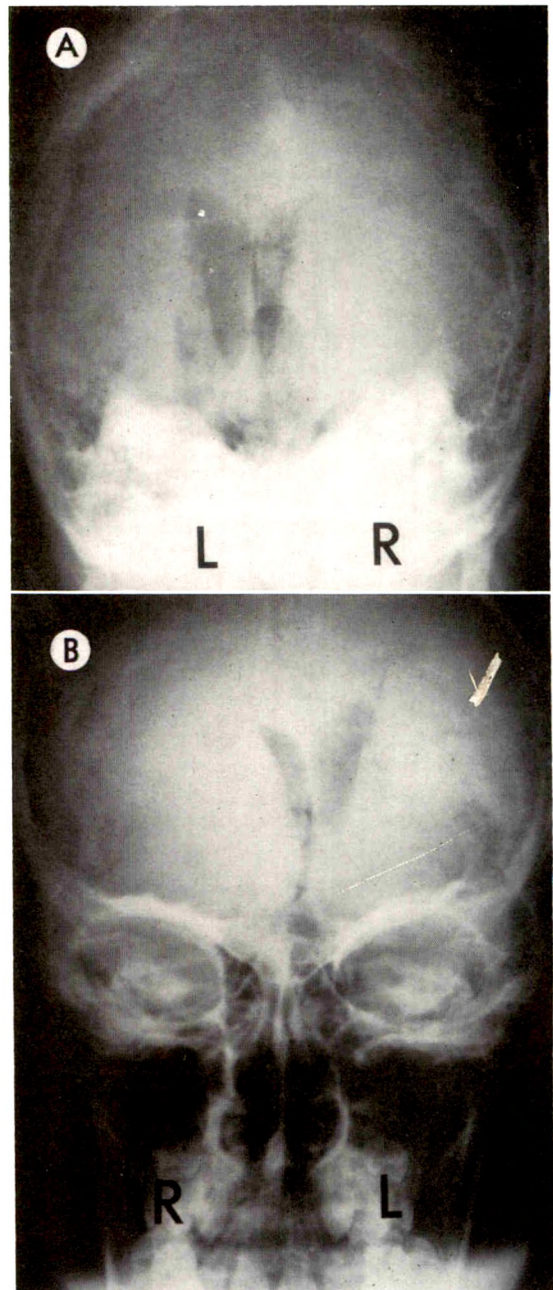


FIG. 14. Case IV. (A) Posterior and (B) anterior pneumoencephalograms made 14 days later reveal shift of midline structures to the left. In addition, there appears to be a mass in the right posterior frontal region.



tetralogy of Fallot was made and a left Blalock procedure was performed.

Following this, he did well over the next 3½ years, until 5 days prior to his present admission, when he developed occipital headaches and a feeling of lethargy.

On admission, his vital signs were: blood pressure 96/30, pulse 56 and irregular, respiration 20/min., and temperature 98.4°F. Physical findings were limited to: cyanosis, clubbing of the extremities and to the cardiac system (a thrill at the left lower sternal border; a Grade III injection systolic murmur in the third left intercostal space transmitted to the right intercostal space; a Grade IV continuous murmur under the left clavicle).

Laboratory findings revealed a white blood cell count of 15,500 with a differential of 6 per cent polymorphonuclear cells, 22 per cent lymphocytes, 7 per cent monocytes, 1 per cent eosinophils and 3 per cent bands. The hematocrit was 49 per cent. Lumbar puncture revealed normal dynamics and a total protein content of 130 mg. per cent. There were 4 white blood cells per high power field.

The electroencephalogram revealed a slow wave focus in the posterior frontal-anterior temporal lobe, consistent with a lesion in this area. The sonar encephalogram showed a 2–3 mm. shift of midline structures to the left.

An Hg<sup>197</sup> brain scan showed localized uptake of isotope superficially in the right posterior frontal lobe (Fig. 12, *A* and *B*).

He was placed on intravenous penicillin (2 million units every 2 hours) and chloramphenicol (400 mg. every 4 hours).

On the 7th hospital day, a carotid arteriogram demonstrated displacement of the anterior cerebral artery beyond the midline to the left and possible distortion of the sylvian vessels. A mass in the region of the posterior frontal lobe was considered possible (Fig. 13, *A* and *B*), but it was felt unlikely that a well localized abscess was, as yet, present.

His clinical condition remained unchanged on antibiotics and on the 31st hospital day pneumoencephalography was performed. This revealed a definite shift of midline structures to the left, more pronounced anteriorly. The findings were interpreted as consistent with a space occupying mass in the right posterior frontal region (Fig. 14, *A* and *B*). Aspiration through a burr hole revealed 35 ml. of purulent material.

His postoperative course was uneventful. Re-

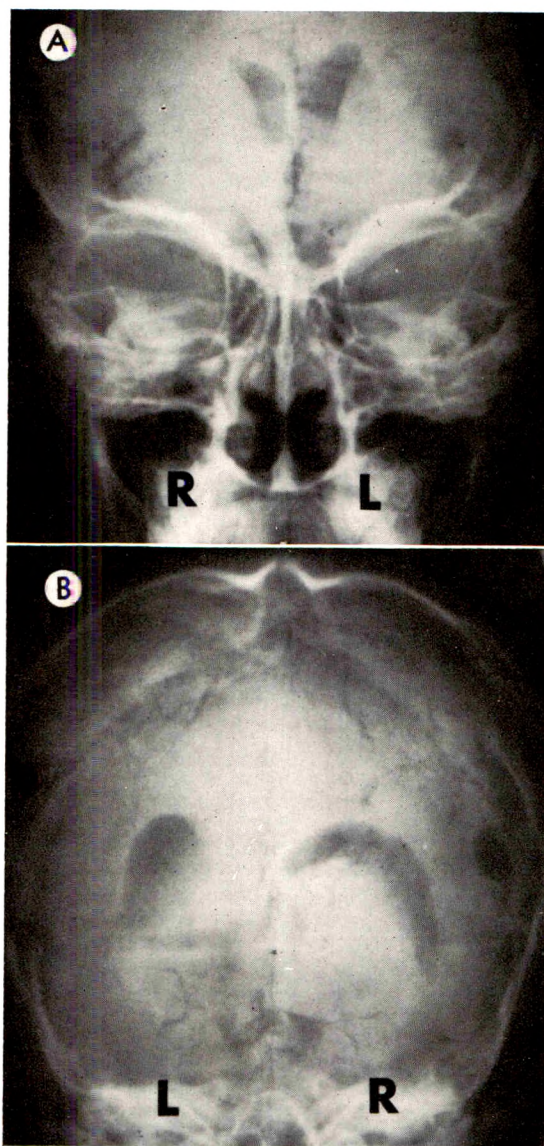


FIG. 15. Case IV. (*A*) Anterior and (*B*) posterior air studies performed on the 54th hospital day show less distortion of the ventricular system.

peat attempted aspirations obtained no additional purulent material. On the 54th hospital day, a repeat air study showed marked improvement with minimal shift of midline structures to the left and less distortion of the right ventricular system (Fig. 15, *A* and *B*). He was then discharged on antibiotic therapy.

#### DISCUSSION

The frequent occurrence of brain abscess in patients with congenital cyanotic heart



disease has been described.<sup>1,3,5,12,13,21</sup> Earlier clinical experience from this hospital has been presented in detail.<sup>8</sup>

Three possible interpretations of the etiology have been discussed:<sup>1,3,12,13,21</sup>

1. Infected emboli are carried across the septal defect and directly into the intracranial circulation.

2. During a transient episode of bacteremia, there may be secondary infection of cerebral thrombi which are frequently present in these patients with polycythemia and decreased cerebral blood flow.

3. There may be infarction of cerebral tissue due to anoxia rather than vascular occlusion; such an area may be susceptible to transient bacteremia with minor infection and a brain abscess may result.

In all of these patients there is a right to left shunt which allows venous blood returning to the heart to enter the cerebral circulation again without previously going through the pulmonary circulation.

The typical clinical history, in this association, begins in a patient invariably more than 2 years of age with cyanotic congenital heart disease. The acute episode may begin with a minor febrile illness,<sup>12</sup> but most often no etiologic factor can be demonstrated.<sup>8,13,21</sup> Seven to 10 days later, the patient may develop headache, vomiting and personality changes. There may be focal neurologic changes and convulsions. Fever may or may not be present.

On laboratory examination, there may be leukocytosis in the peripheral blood but an increased polymorphonuclear cell count may not be noted. Polycythemia in such patients with elevation of the hematocrit and hemoglobin is usual. Lumbar puncture usually shows an increased cerebrospinal fluid protein content, the sugar content is normal, and the cell count may or may not be increased. Positive blood cultures are often not obtained.<sup>8</sup> Indeed, under antibiotic therapy, at least 25 per cent of cases will have sterile cultures on aspiration of the abscess cavity.<sup>8,12</sup>

Notoriously, these abscesses have been subtle in origin, difficult to prove in the

early phases and accompanied by a high morbidity and mortality because of delay in recognition and proper treatment.

In a review of the literature concerned with the radioisotope brain scan, several authors have noted their limited experience in patients with brain abscess.<sup>2,4,10,14,17</sup> Description of the clinical situations is not given in these cases. In one paper,<sup>4</sup> it is mentioned that 2 patients with a positive brain scan also had positive studies by cerebral arteriography, but the time sequence between these studies is not given.

In our own recent limited experience, certain features of special interest seemed worth presenting, possibly to improve the frequency of early detection and better management of these difficult lesions. These features are perhaps best illustrated by Case I. One notes that certain physiologic tests such as the electroencephalogram and the radioisotope brain scan may show definite abnormality before such anatomic studies as the cerebral pneumoencephalogram and arteriogram. That is, investigative procedures which detect an abnormality of function in a given area of brain may be positive considerably earlier than those which show only an abnormality of structure, *i.e.*, the presence of a mass. Thus, in Case I, the physiologic tests were abnormal possibly at a time of "cerebritis" and before actual liquefaction of tissue occurred with resultant formation of an abscess cavity. Three weeks later, as the brain scan remained definitely positive, air study became positive and an abscess cavity was found at surgery. The same time relationships between positive physiologic tests (*i.e.*, electroencephalogram and isotope scan) and the anatomic roentgenographic procedures (*i.e.*, air study and cerebral arteriogram) in the early and late stages of the patient's course were also noted in Case III and perhaps also in Case IV to a lesser degree.

One might compare this situation with the experience of others in the case of encephalomalacia.<sup>7,11,15,19,22,23</sup> With cerebral infarction, the isotope scan is positive within the first few weeks but later reverts to

normal, while the arteriograms may remain within normal limits at all times throughout the clinical course. Such a situation is said to differentiate a solid tumor, where the brain scan would be just as positive in a late as an earlier phase, from the condition of encephalomalacia.

In both, the instance of cerebritis and of encephalomalacia, the initially positive brain scan points to a breakdown of the normal blood-brain barrier. Later, as the cerebritis becomes more "localized" with formation of an actual abscess cavity, a localized lesion or "mass" can be demonstrated unequivocally by an air study, whereas with encephalomalacia no mass develops, and one would expect complete resolution by all studies. If a true cerebritis without resultant abscess formation could be encountered, one would expect the sequence not to be unlike that of encephalomalacia. Although a brain abscess in the cyanotic heart disease patients under discussion is usually solitary,<sup>3,13</sup> multiple abscesses were found at autopsy in Case II. Only one abscess was noted by the radioisotope scan and electroencephalogram. We would agree, therefore, with the experience of others<sup>2,15</sup> that the brain scan may not be completely reliable, although there has not been a study in a large number of patients to evaluate this accurately. However, we feel that in the patient with cyanotic congenital heart disease, who may be suspected of having a brain abscess, the radioisotope scan is a relatively simple and safe method which can be used as a screening procedure. It may localize the lesion well before the more involved roentgenographic contrast examinations and at a time when clinical examination is not definitive. With an isotope of even lower radiation dosage than we have used, such as Tc<sup>99m</sup> multiple serial scanning may prove even more profitable in evaluating spread or localization of the process from cerebritis to abscess formation.<sup>9,16,24</sup>

#### SUMMARY

Four children with cyanotic congenital heart disease, who were studied for the complication of brain abscess, are described.

An earlier stage of "cerebritis" is compared to the later stage of localized suppuration in relation to the finding of 2 types of radiographic procedures: the isotope brain scan as a test of function and air encephalography or arteriography as a test of structural alteration.

The importance of these studies in planning the clinical management of these patients is discussed. It is noted that an initially positive brain scan may occur at a time when the lesion is not yet suitable to surgical drainage. It appears that such drainage had best be delayed until the test of structure, the contrast roentgenographic procedures, also indicate a localized mass lesion.

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We wish to acknowledge our thanks to Dr. Paul J. New for reviewing the air studies in Cases I, III and IV and the arteriogram of Case IV.

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## ROENTGENOGRAPHIC MANIFESTATIONS OF COMPLICATIONS SECONDARY TO CEREBRAL VENTRICULAR- CARDIAC ATRIAL SHUNTS

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THE most favorably regarded treatment for infantile hydrocephalus is presently considered to be the establishment of a shunt from the cerebral lateral ventricle to the cardiac right atrium, incorporating a unidirectional valve mechanism.<sup>5,17</sup> As of 1960 an estimated 5,000 valves had been inserted.<sup>24</sup> A more recent report indicated 550 patients treated in one institution.<sup>29</sup>

The concept of decompressing hydrocephalus with a shunt into the vascular bed was first suggested by Gartner at the National Science Convention of 1895 in Lübeck. Subsequently, in 1908 Payre reported 3 cases in which he anastomosed the lateral ventricle to the longitudinal sinus with an autogenous vein graft. The following year McClure<sup>18</sup> described a shunt from the dura to the external jugular vein. These and other early vascular shunt procedures have been well reviewed.<sup>4,13,23</sup> In general, the results were poor due to occlusion by collapse or clotting of blood.

In a controlled experimental evaluation, Ingraham *et al.*<sup>11</sup> utilized dogs in which they had induced obstructive hydrocephalus. While the animals were under anesthesia, the pressure gradient was favorable for the flow of cerebrospinal fluid to the venous system, even after the initial high intracerebral pressure had been reduced. However, in the awake and active dog, the episodic strain of excitement and coughing was found to raise the venous pressure sufficiently to cause backflow of blood into the system which would then clot. They concluded that ventriculo-venous shunts would fail unless an "extremely sensitive but completely competent artificial valve"

could be interposed into the system to allow only unidirectional flow.

This was accomplished by Nulsen and Spitz<sup>21</sup> by the use of a double stainless steel ball valve device. Their report in 1951 described a patient having such a valve in place for 2½ years with the ventricular pressure normal and the system functioning. The valve mechanism itself was located subcutaneously over the mastoid with the catheters attached to either end. Pudenz and co-workers<sup>23</sup> subsequently developed a valve which was incorporated into the end of the tubing and bathed in the blood of the right atrium.

The currently available Spitz-Holter valve, used since 1956, is a modification of the Nulsen-Spitz variety with a double fishmouth valve mechanism. The unit is cylindrical, measuring 5 cm. in length and 0.6 cm. in diameter. The catheters are composed of siliconized rubber impregnated with 5 per cent barium sulfate to allow roentgenographic visualization (Fig. 1).

The inherent advantages of ventriculo-atrial shunts include the following: (1) the same procedure may apply to obstructive or communicating hydrocephalus; (2) cerebrospinal fluid is retained; (3) a normal organ is not sacrificed (as opposed to nephrectomy for uretero-arachnoid anastomoses);<sup>16</sup> (4) the system is closed, obviating the risk of ascending infection; and (5) the operative procedure is relatively simple.

These shunts should not be instituted in the presence of central nervous system infection, residual intraventricular air from previous ventriculography or pneumoencephalography, or when the cardiac status is such that the right atrial pressure is



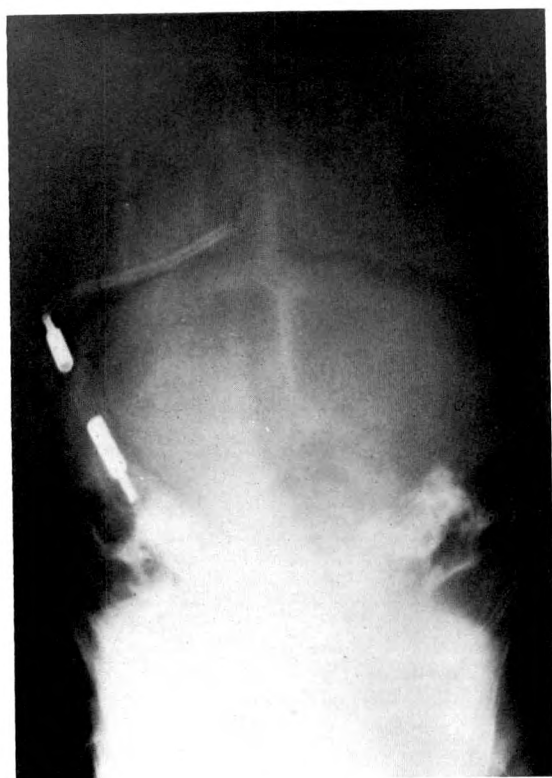


FIG. 1. Towne projection. A Spitz-Holter valve is present on the right. The intraventricular catheter is well positioned within the right lateral ventricle.

elevated to a degree which will not allow effective decompression of the cerebral ventricles (Spitz-Holter valves are designed to open at either 10 or 50 mm. of water pressure).

The currently available valves may be tested percutaneously by direct pressure on the valve device itself (Spitz-Holter), or an accessory pump (Pudenz-Heyer). The cardiac tips should be placed within the right atrium or at the junction of the right atrium and the superior vena cava. Placement within the superior vena cava or jugular vein will frequently lead to occlusion by fibrinous deposits.<sup>1,22</sup>

#### ROENTGENOLOGIC ASPECTS

##### CATHETER POSITION

Preliminary chest roentgenograms including the neck and lateral skull are obtained to estimate the length of catheter

needed. This should then be verified at the time of surgery by a repeat roentgenogram or with image intensification equipment. Electrocardiographic monitoring has also been used to establish the position of the cardiac catheter tip.<sup>25</sup>

#### COMPLICATIONS

Surgical revision or replacement is necessary in approximately 20–25 per cent of the cases.<sup>19,20,26</sup> The main indications are sepsis, thromboemboli, body growth, and malfunction.

1. *Sepsis and Thromboemboli.* Sepsis occurs at a rate estimated at 15–20 per cent and is responsible for the majority of early deaths.<sup>20</sup> A syndrome of pyrexia, splenomegaly, and progressive anemia with positive blood cultures may develop subacutely. This is a manifestation of colonization of the valve by organisms of relatively low virulence.<sup>2</sup>

Thromboemboli are frequently associated with sepsis, and are present in a distressingly high incidence of all cases examined pathologically. Friedman *et al.*<sup>8</sup> reviewed the findings of 65 patients who died with ventriculovascular shunts. Various thromboembolic phenomena were observed in 57

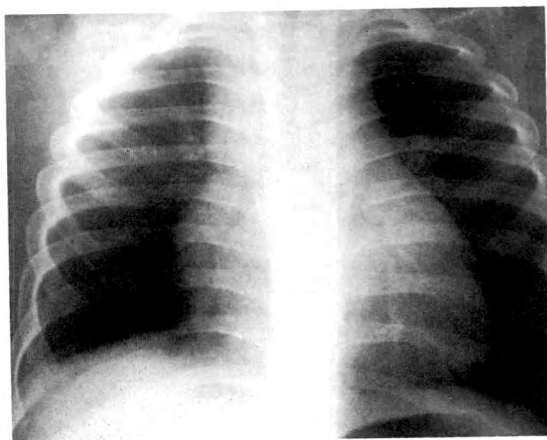


FIG. 2. This 3 year old male developed intractable congestive failure several months after a shunt revision. Multiple thromboemboli were present within the lungs at the time of postmortem examination. Cardiomegaly, small right pleural effusion, and evidence of pulmonary hypertension may be noted on this chest roentgenogram.

per cent. This was nearly twice the frequency found in a group of controls, consisting of patients who died with hydrocephalus but did not have shunt procedures as their therapy. In another autopsy series,<sup>6</sup> all 15 patients had a sleeve of fibrinous material surrounding the catheter in the jugular area, which in some instances extended into the right atrium. Emboli or thrombi were present in the pulmonary circulation of 14 of the cases. In 2 instances, massive emboli were felt to be the immediate cause of death. Atrial mural thrombi were also identified, and have been detected angiographically as well as pathologically.<sup>27,28</sup> They may alter the function of the tricuspid valve, embolize, or colonize to form a bacterial endocarditis.

Cor pulmonale, both acute and chronic, has been reported in postshunt patients by Noonan and Ehmke.<sup>20</sup> They feel that the majority of those dying with acute sepsis have septic emboli and terminal acute cor pulmonale (Fig. 2).

2. *Growth.* Body growth itself may necessitate revision because of proximal retraction of the cardiac catheter into the superior vena cava or internal jugular vein, where occlusion by thrombosis is probable. The average growth of a child from the mastoid tip of the xiphoid averages only 6 cm. during the first 5 years of life.<sup>23</sup> Therefore, if some allowance is made in placement within the right atrial chamber, these revisions need not be done frequently.

The original hope—largely based on Laurence's<sup>14</sup> report of 45.5 per cent spontaneous arrests in untreated patients—that arrest of the underlying hydrocephalus would frequently obviate the need for revision has not as yet been realized. Some feel that the presence of the shunt may remove a stimulus for development of compensatory pathways of cerebrospinal fluid absorption, and that functioning shunts will be necessary throughout the entire life of the patient.<sup>7</sup>

3. *Malfunction of Valve Mechanism.* Occlusion of the system may occur as a result of thrombus formation, brain tissue plug,

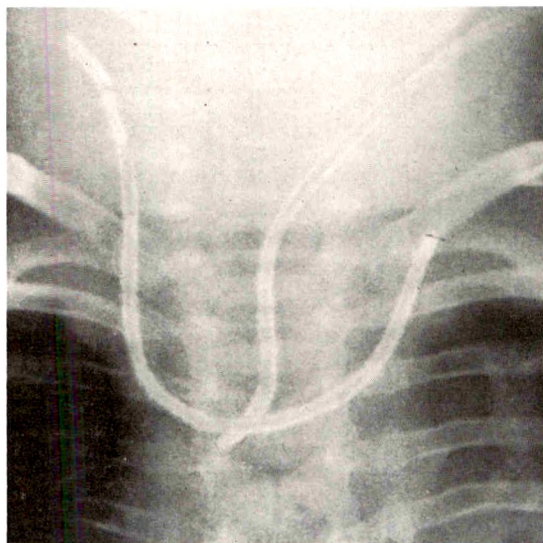


FIG. 3. This 1 year old male has had multiple revisions of his shunt, including the placement of a second Spitz-Holter valve on the left. This chest roentgenogram demonstrates the tip of the cardiac catheter from the left-sided valve in the region of the left innominate vein. In the lower cervical region it is more medial than the internal jugular vein, entering the left innominate vein through a parallel channel. The catheter from the right-sided valve has migrated through the left innominate vein to the left subclavian. (Catheters retouched to enhance visualization.)

and separation or failure of the component parts of the mechanism. If the location is proper and the continuity maintained, the main roentgenographic evidence of occlusion is that of increasing intracranial pressure. Pseudocysts containing cerebrospinal fluid also develop about the valve and tube tracts, secondary to obstruction or leakage from connecting points.

The cardiac catheter may be misplaced or migrate into an undesired vessel such as the subclavian, innominate, or jugular veins (Fig. 3). It may completely separate and lodge in the pulmonary arteries,<sup>8</sup> or right cardiac chambers (Fig. 4). This has been demonstrated angiographically,<sup>3</sup> but should be discernible on good quality plain roentgenograms when radiopaque catheters are used.<sup>10</sup>

There have been reports of perforation of the right atrium<sup>5,29</sup> and even the right ven-



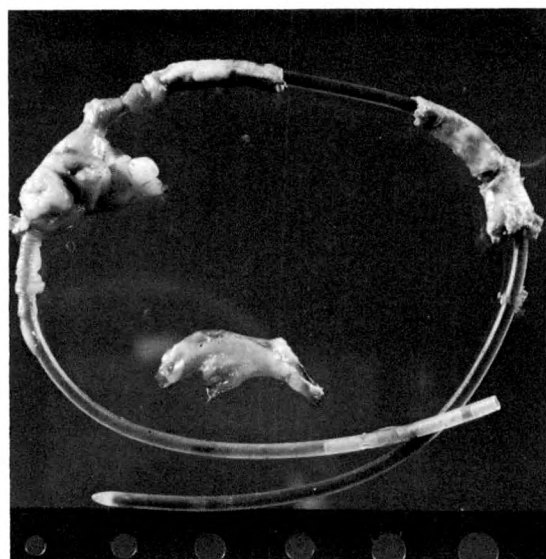


FIG. 4. A Pudenz-Heyer tube with adherent fibrinous material had separated and lodged predominantly in the right atrium. It was surgically removed.

tricle<sup>27</sup> by the cardiac catheter tip. Pericardial effusion and cardiac tamponade have resulted. Additional complications may result from rapid decompression of a particularly large hydrocephalus. These include the development of subdural hematomata secondary to tearing of bridging veins, epi-

dural bleeding,<sup>15</sup> and circulatory overload with congestive failure. In 1 instance craniosynostosis occurred after the parietal bones approximated.<sup>27</sup>

The following case illustrates another unusual complication.

#### ILLUSTRATIVE CASE

R.B., a male infant, had a Holter valve inserted at 2½ months of age. This functioned satisfactorily for the succeeding 7½ months, at which time considerable interstitial fluid accumulated in the upper cervical region and the cardiac tube had retracted, necessitating a revision.

This was accomplished by interposing an extension tube between the valve and cardiac catheter to restore its length. On the fifth postoperative day, the patient developed a pleural effusion and right upper lobe atelectasis (Fig. 5*A*). He was afebrile and had a normal white blood cell count. Because of the possibility that this effusion might represent cerebrospinal fluid, a thoracentesis was performed 1 hour after the intraventricular instillation of 1.5 cc. of indigo carmine and manual pumping of the valve mechanism. The pleural fluid was initially clear, then became blood tinged; but no dye was identified. Subsequently, the fluid

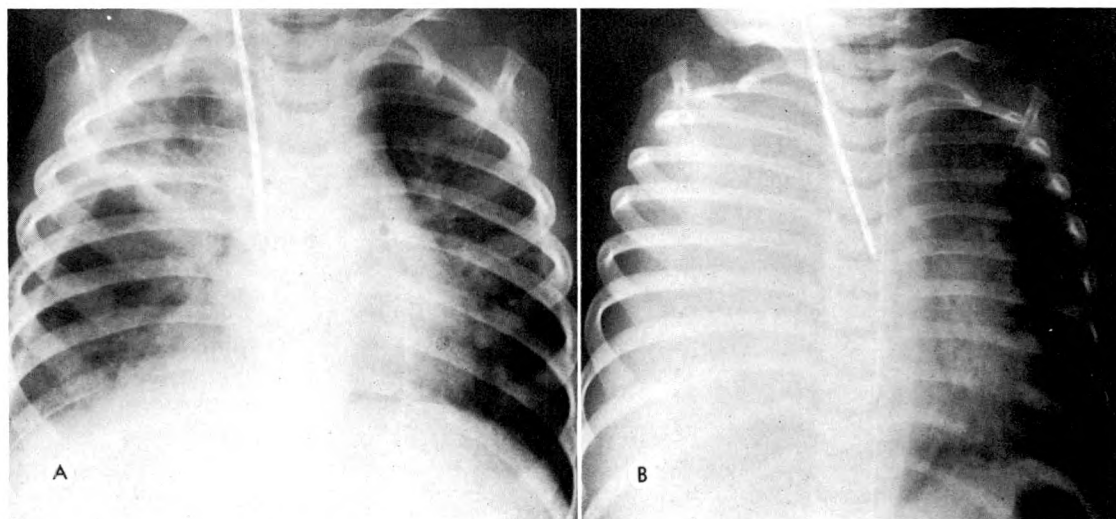


FIG. 5. (*A*) Right upper lobe atelectasis and a right pleural effusion are apparent. The cardiac catheter as visualized on this chest roentgenogram is in the desired position. (*B*) A chest roentgenogram obtained 2 months later demonstrates a massive right pleural effusion with displacement of the mediastinal structures toward the left. (Catheter retouched to enhance visualization.)

and atelectasis seemed to be resolving and the patient was discharged.

He returned 2 months later with a history of progressive dyspnea leading to cyanosis. A massive right pleural effusion with displacement of the mediastinal structures toward the left was apparent on the chest roentgenogram obtained at that time (Fig. 5B). The cardiac catheter was likewise displaced. Again, a thoracentesis was performed following instillation of indigo carmine via a ventricular puncture. At this time, blue tinged fluid was obtained from the pleural cavity, demonstrating a communication. An operative procedure was done in which the cardiac catheter was withdrawn and a ventriculo-peritoneal shunt established.

*Comment.* Ventriculo-pleural shunts had been performed in the past, but abandoned because of the necessity of repeated surgical procedures.<sup>13</sup> Ingraham *et al.*,<sup>12</sup> however, demonstrated that dogs are able to absorb normal saline through the pleural surface. In this patient the reason for increasing accumulation is not known. It may have been related to the presence of blood in the pleural cavity noted at the time of the initial thoracentesis.

#### SUMMARY AND CONCLUSION

The roentgenographic evaluation of post-operative ventriculo-atrial shunt patients should include examinations of the skull, neck, and chest. Comparison with previous roentgenograms is necessary for early detection of complications and to assess the effect of growth. Specifically, the following are recommended:

1. Identify the valve mechanism and catheters, and ascertain their position and continuity.
2. Note evidence of increasing intracranial pressure.
3. Think of thromboemboli and carefully scrutinize the chest roentgenogram for signs of pulmonary hypertension, pulmonary infarction, and congestive failure.
4. Utilize angiocardiology when necessary to confirm the presence of mural thrombi, pulmonary vascular changes, and pericardial effusion.

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## ROENTGENOLOGIC FINDINGS IN SYRINGOMYELIA AND HYDROMYELIA\*

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THE term syringomyelia may be used in two senses. It may be used to describe a clinical syndrome characterized by atrophy in the upper extremities, spasticity of the lower extremities, dissociated or "suspended" sensory deficit and scoliosis. It may also be used to describe a pathologic condition characterized by gliosis and longitudinal cavitation of the spinal cord. The term hydromyelia refers to a condition in which some or all of the central canal of the spinal cord is expanded by fluid and may communicate with the 4th ventricle at the obex. There is some confusion about the words syringomyelia and hydromyelia. Some authors use the word syringomyelia for both conditions. Others use the word hydromyelia for a large syringomyelic cavity. In any case, it is usually impossible to determine before autopsy whether or not a fluid filled cavity in the spinal cord is the enlarged central canal.

In hydromyelia the spinal cord is usually a large thin-walled tube filled with fluid, the fluid usually being clear and of low protein content. Occasionally, the spinal cord is of average size. Under certain conditions, it may appear to be small. Hydromyelia represents persistence of the normal fetal condition in which the 4th ventricle communicates with the central canal of the neural tube. Sometimes, only part of the central canal is enlarged. It is likely that hydromyelia itself gives rise to few or no symptoms, any symptoms or signs being due to accompanying anomalies such as Arnold-Chiari malformation, hydrocephalus, etc.

In syringomyelia, the appearance of the spinal cord varies. If the syringomyelic cavities are microscopic, the spinal cord is usually small or average in size. If the syringomyelic cavity or cavities are large,

the spinal cord may be large. This enlargement can be either fusiform or tubular, depending on the length of the syringomyelic cavity or cavities.

The spinal cord is probably distended with fluid in childhood in most cases of syringomyelia and hydromyelia. However, in some adults the spinal cord is found to be small. This must be due to absorption of fluid from the cavity in some cases and due to communication with the 4th ventricle or subarachnoid space in other cases.

Syringomyelia may be primary or secondary. Primary syringomyelia is a disease of unknown etiology occurring usually in young adults. The condition is probably less common than has been thought in the past. Certain cases considered to be childhood syringomyelia were almost surely examples of hereditary neuropathy<sup>4</sup> and others may have been cases of diastematomyelia. The term syringomyelia has been used in the past to describe cavities in the spinal cord secondary to a great variety of diseases. The use of the word syringomyelia in such cases does not help us to understand the pathologic processes or the clinical course of the disease. It may even tend to obscure the true nature of the cavities in the cord or the symptom complex. We hope that the term syringomyelia will be used less and less and that terms such as post-traumatic gliosis and cavitation of the spinal cord will be used more and more.

Lesions that may produce secondary syringomyelia or which may be found in patients with syringomyelic-like syndromes can be grouped as follows:

Developmental:

Dysraphism, presumably with ependymal rests in the cord

Bone anomalies at the foramen magnum

\* Presented at the Second Annual Meeting of The American Society of Neuroradiology, Atlantic City, June 11, 1965.



Anomalies of the upper cervical vertebrae  
 Anomalies of other vertebrae, including diastematomyelia  
 Arnold-Chiari malformation  
 Traumatic:  
   Intramedullary hematoma  
 Vascular:  
   Infarction of the cord  
   Angioma of the spinal cord  
 Inflammatory:  
   Virus myelitis  
   Bacterial meningitis and myelitis  
 Neoplastic:  
   Glioma with cyst formation  
 Degenerative:  
   Cervical spondylosis

The roentgenologic findings depend on the type of lesion in the cord as well as the etiology. Ordinary spine roentgenograms may show:

Large spinal canal:  
   Upper cervical region  
   Cervical and/or thoracic region  
 Bone anomaly:  
   Foramen magnum  
   Upper cervical vertebrae  
   Other vertebrae  
 Old fracture or fracture dislocation  
 Old osteomyelitis or tuberculosis  
 Cervical spondylosis  
 Scoliosis or kyphoscoliosis

Gas and/or oil myelograms may show one or more of the following findings in the same patient:

Large spinal cord  
 Small spinal cord  
 Collapsing spinal cord  
 Subarachnoid adhesions  
 Arnold-Chiari malformation

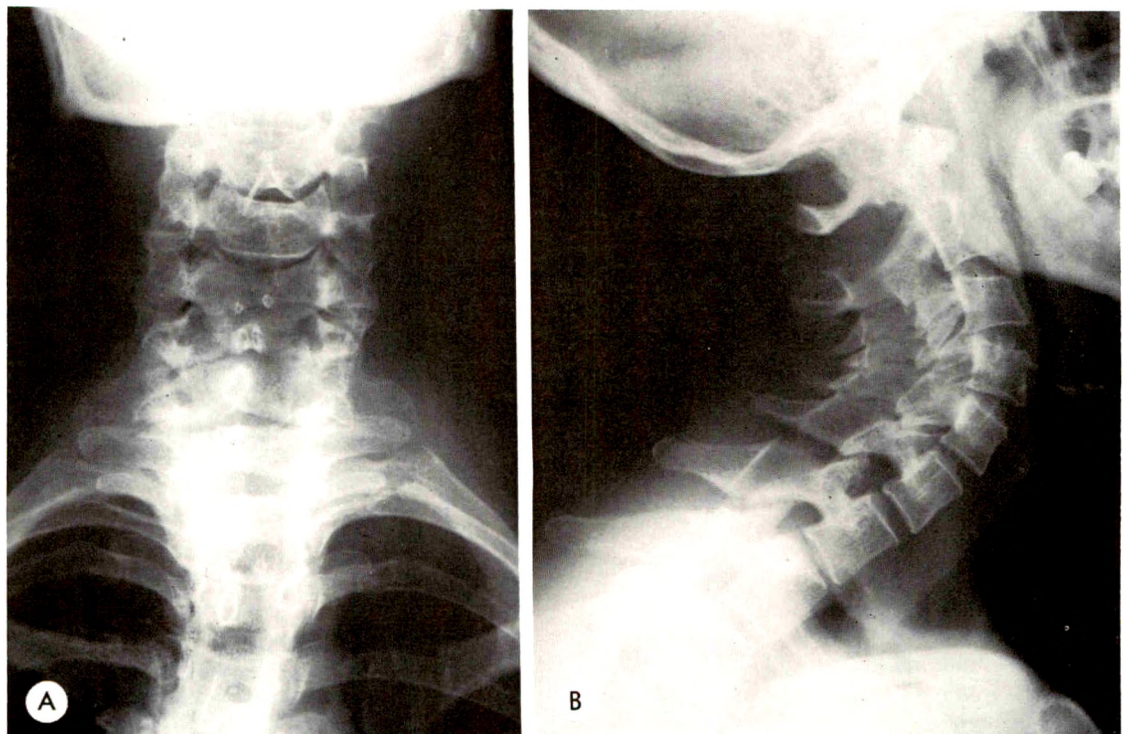


FIG. 1. (A and B) M. G., female, aged 41 years, showing increased width and depth of the cervical spinal canal. She had had dull aching pain along the lateral surface of the right arm for 10 or 12 years with some weakness and stiffness of the right hand, mainly involving the fourth and fifth fingers. She had moderate to marked dorsal kyphosis but no scoliosis. There was loss of stereognosis of the right third, fourth and fifth fingers, diminished temperature sensitivity in the right forearm, diminished touch sensitivity in the right ring finger and slight smallness of the right forearm but no noticeable atrophy of the right hand. The biceps reflexes were absent in both arms but otherwise the deep tendon reflexes were normal. At operation, a large fluid-filled cervical cord was found from which 3 cc. of slightly yellow high-protein fluid was withdrawn.

TABLE I  
SPINAL CANAL IN SYRINGOMYELIA AND HYDROMYELIA

		Transverse Diameter	Anteroposterior Diameter	Both Increased
Proved (20 cases)	Normal	10*	11*	7
	Borderline	5	2	
	Definite	4	6	
Unproved (33 cases)	Normal	23*	26*	6
	Borderline	5	2	
	Definite	4	4	

\* One patient had no cervical spine studies.

The case records of the Montreal Neurological Institute from June, 1934 to December 31, 1964 were searched and 55 patients were found in whom the diagnosis of syringomyelia had been made. Study of the case records showed that in 20 of the 55 there was surgical and/or postmortem confirmation of the diagnosis. In 2 patients, postmortem examination disproved the diagnosis. None of the remaining 33 was operated on nor was autopsy carried out.

ROENTGENOLOGIC FINDINGS

The cervical spinal canal was often large in syringomyelia and hydromyelia (Fig. 1, A and B). In some cases it was the anteroposterior diameter which was increased, in other cases it was the transverse diameter, while in still others both diameters were increased. The transverse diameter of the spinal canal was measured and compared with Dyke and Elsberg's table.<sup>2</sup> The anteroposterior diameter of the cervical spinal canal was measured and compared with Burrows' table<sup>1</sup> and/or Hinck's table.<sup>5</sup> Ten of our 20 proven cases of syringomyelia and hydromyelia had enlargement of the cervical spinal canal in either anteroposterior or transverse diameter and in 7 both diameters were increased (Table I). In 7, the enlargement was moderate or marked and in 3 it was slight. The walls of the spinal canal were not decalcified, indicating that the enlargement had been present for life or else had occurred very slowly. Three of the

10 cases had slightly larger thoracic spinal canals, mainly in the upper thoracic region.

Before roentgenograms of the head or neck were made, the clinical diagnosis of syringomyelia was often suggested in patients who had bone anomalies at the cranio-vertebral junction.<sup>7</sup> Operation and/or postmortem examination disclosed syringomyelia and/or hydromyelia in less than half of such cases, however. In most patients with symptom-producing occipitalization of the atlas or atlanto-axial dislocation, there was localized atrophy of the upper part of the spinal cord at the level of the odontoid process. Below this level, the spinal cord was often normal in appearance. In a few cases it was found to be small but it was impossible to say, except at autopsy, whether this was simple atrophy of the spinal cord or whether it represented syringomyelia or hydromyelia. Bone anomalies in the occipito-cervical region were present in 4 of our 20 proven cases of syringomyelia and hydromyelia. When the bone anomaly was accompanied by a true Arnold-Chiari malformation, there was apt to be hydromyelia (Fig. 2).

Other types of bone anomalies of the spine were rare in our series, although they were rather common in Gardner's<sup>3</sup> series. Diastatomyelia produces neurologic syndromes sometimes diagnosed as thoracic or lumbar syringomyelia before roentgenologic examination is carried out. In such cases, the spinal cord changes should prob-



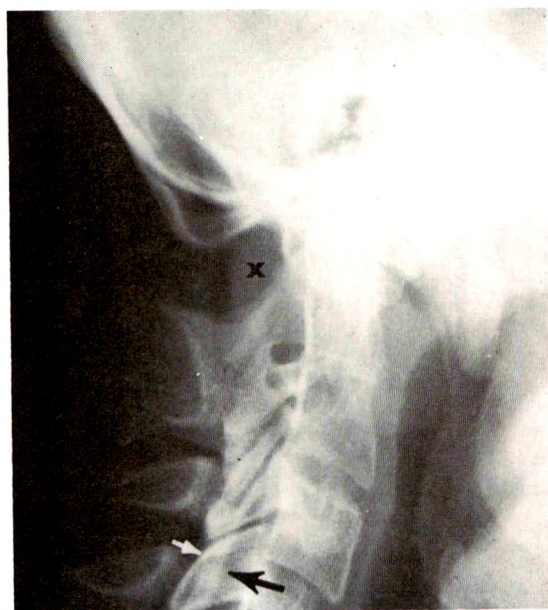


FIG. 2. P. O., male, aged 22 years, with occipitalization of the atlas and an Arnold-Chiari malformation (x). The upper cervical cord is seen to be small on the gas myelogram (arrows).

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ably not be called syringomyelia and, in fact, at autopsy syringomyelic changes are rarely present.

Evidence of old fracture or fracture dislocation of the cervical spine was seen in 1 of our 20 proven cases (Fig. 3, *A* and *B*).

None of our proven cases showed evidence of old spinal osteomyelitis or tuberculosis, but such cases have been reported and we have recently seen one. This patient had had thoracic Pott's disease. Some years later slowly progressive paraplegia devel-

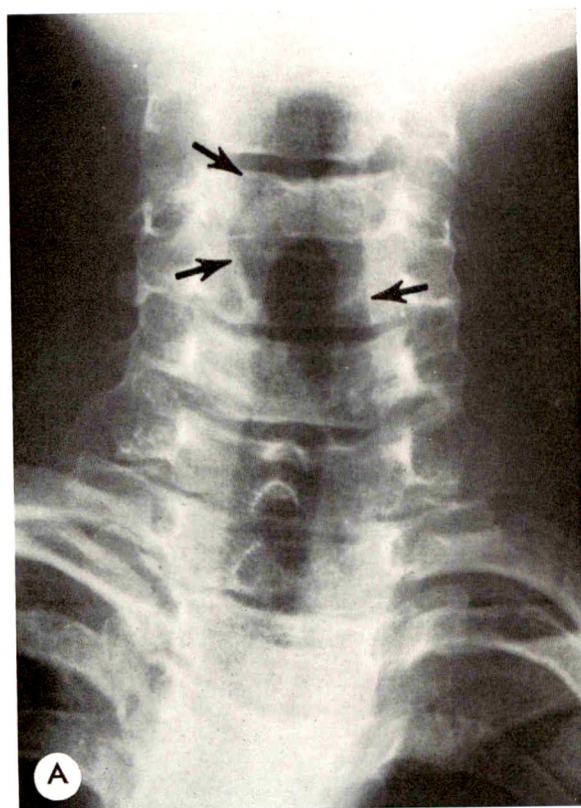


FIG. 3. (*A* and *B*) P. L., male, aged 11 years, with a large triangular cyst in the cervical spinal cord filled with air. At the age of 4 years, he was said to have had a fracture-dislocation of the cervical spine which had to be reduced surgically.



TABLE II  
BONE CHANGES IN SYRINGOMYELIA AND HYDROMYELIA

	Gradation	Proved (20 cases)	Unproved (33 cases)	Disproved (2 cases)
Bone anomaly about foramen magnum		4/19*	4/32*	0/2
Bone abnormality of cervical or thoracic spine		4/19*	2/32*	0/2
Enlargement of cervical and/or thoracic spinal canal	Borderline	3	4	0
	Definite	7	5	0
	Total	10/19*	9/32*	0/2
Cervical and/or thoracic scoliosis	Slight	5	14	0
	Definite	12	15	0
	Total	17/20	29/32*	0/2

\* One patient had no cervical spine studies.

oped and there was a curious asymmetric sensory loss. The myelogram showed arachnoiditis at the level of the old bone disease. Operation disclosed intra- and extradural adhesions and a soft fluctuant spinal cord below the level of the arachnoiditis. Yellow high protein fluid was aspirated from the cord here.

Cervical spondylosis is so common in middle aged and elderly patients that it is difficult to assess its neurologic significance. In the past, some patients with cervical myelopathy secondary to cervical spondylosis (discogenetic disease) were considered as having syringomyelia. However, the spinal cord in these patients rarely shows cavitation and if such were found the association might be a chance one.

Scoliosis or kyphoscoliosis, either cervical or thoracic, was present in 17 of our 20 proven cases. It is one of the classic signs of syringomyelia and probably is due to imbalance of muscle tone in the right and left erector spinae muscles. Muscle imbalance is probably the cause of the increased cervical lordosis frequently noted.

The bone abnormalities found in our cases are summarized in Table II.

The myelographic findings were variable since the fluid-filled spinal cord varied in

size with the position of the patient, with the presence or absence of communication between the cavity and the 4th ventricle or subarachnoid space and with the presence or absence of fluid around the cord. When

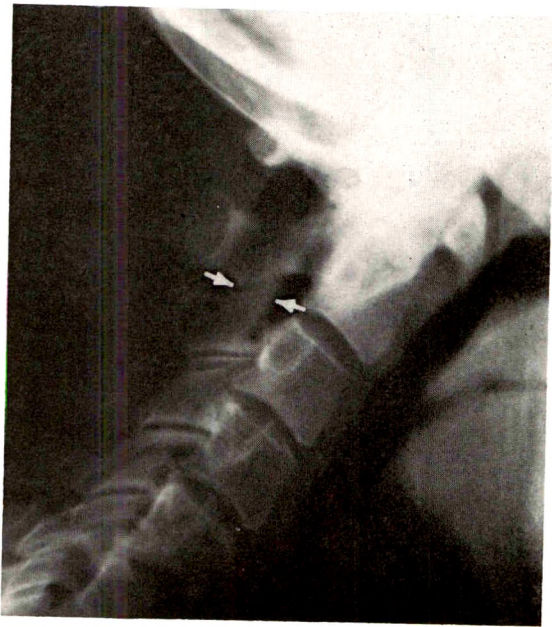


FIG. 4. M. H., male, aged 46 years. Gas myelogram with the patient erect showing a small cervical spinal cord shadow. (Reproduced with permission from *Rad. Clin. N. America*, 1966, 4, 145, W. B. Saunders Co., Publishers, Philadelphia, Pa.)



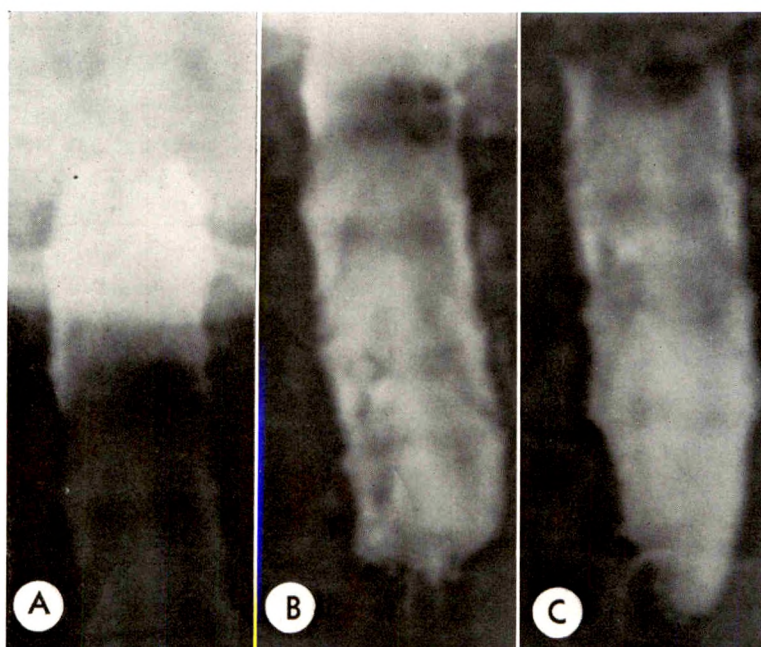


FIG. 5. (A, B and C) Same patient as in Figure 4. Oil myelograms with the patient prone and the roentgenographic table horizontal showing a large cervical spinal cord shadow. (Reproduced with permission from *Radiol. Clin. N. America*, 1966, 4, 146, W. B. Saunders Co., Publishers, Philadelphia, Pa.)

the patient was upright and the supporting cerebrospinal fluid was removed, as in gas myelography, the fluid inside the spinal

cord tended to run downwards (Fig. 4). The upper part of the saccular cord then collapsed and was seen as a small cervical cord shadow on the gas myelogram. When the patient was horizontal (Fig. 5, A, B and C), the fluid in the cord ran back up into the cervical cord, causing an enlarged cervical cord shadow on the oil myelogram. If the cavity in the cord was long, the fluid in the cervical cord passed into the thoracic cord when the patient was upright, causing a small cervical cord shadow throughout its full length (Fig. 6). If the cavity in the cord was short and the patient erect for the gas myelogram, the upper part of the cord appeared small while the lower part appeared large (Fig. 7). None of our cases



FIG. 6. D. B., male, aged 30 years, complaining of progressive weakness and stiffness of the right hand for 2 years. There had also been numbness and tingling of the left hand for 6 months. The gas myelogram with the patient erect shows a small spinal cord, down to C 6 at least.



FIG. 7. M. P. C., female, aged 30 years, complaining of right ptosis and diplopia with pain in the right arm and chest for the past 8 years. There was diminished sensation in the right arm and right thorax. The gas myelogram with the patient erect shows a small spinal cord, down to the C1-C2 level. Below this, the cord enlarges somewhat down to the subarachnoid fluid level at the lamina of C2.



had manometric block or myelographic block. The cord shadow sometimes appeared large, yet operation showed it to be small. At other times, the cord shadow was small, yet operation revealed a large cord. This could be due to myelography being done in a different position than operation. It could be due to the dentate ligaments keeping the cord more or less normal in width but not keeping it from collapsing from front to back (Fig. 8, *A* and *B*; and 9, *A*

and *B*). Possibly, the pressure of heavy iodized oils can flatten a very thin fluid-filled spinal cord. The myelographic findings are summarized in Table III.

Tables I, II and III require some explanation.



FIG. 8. (*A* and *B*) G. B., male, aged 37 years, with weakness and atrophy of the arms for approximately 8 years, along with some weakness and spasticity of the legs. There was a dissociated sensory loss extending from C2 to C8. The lateral cervical roentgenogram shows a large spinal canal and the erect lateral gas myelogram (tomogram) shows a thin ribbon-like cervical cord (arrows).



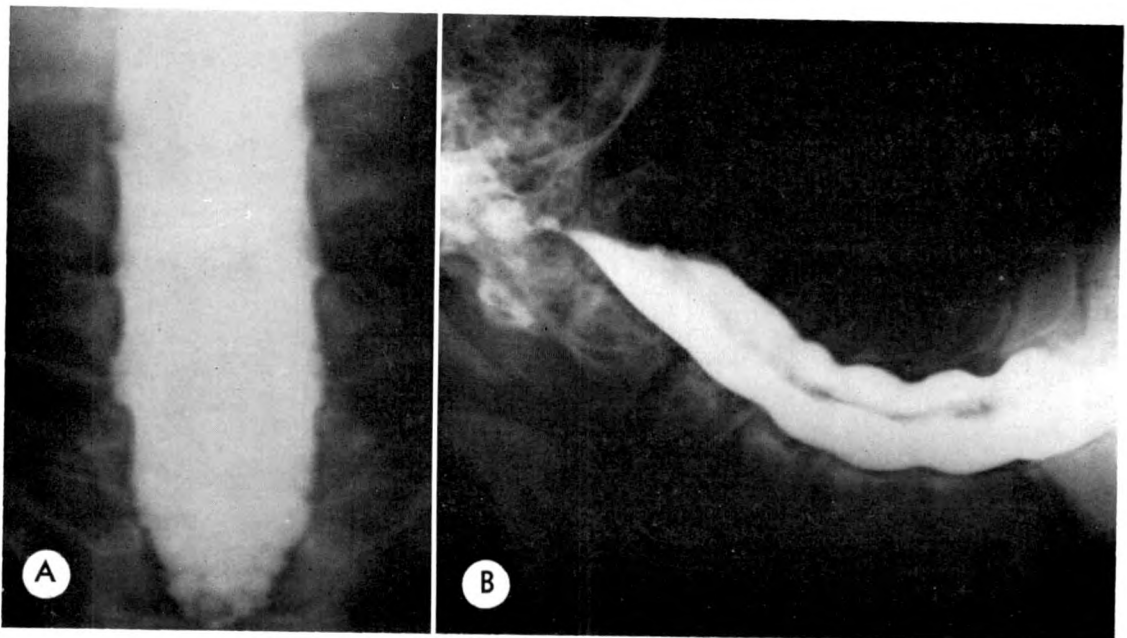


FIG. 9. (A and B) Same patient as in Figure 8, A and B. Oil myelograms on the same date showing a thin ribbon-like spinal cord. Laminectomy was performed 10 days after myelography at the C1, C2 and C3 levels with the patient erect. The upper cervical cord was found to be a thin walled tube, distended and with fluid filling the spinal canal completely. The cause of the discrepancy was not obvious. The fourth ventricle was not completely visualized but no fluid came out of the floor of the fourth ventricle when the spinal cord was milked upwards; therefore, we assume that the obex was closed.

tion. The small number of cases showing bone anomalies at the foramen magnum, atlas and axis is due to the fact that in recent years we have not used the diagnosis "syringomyelia" in such cases. We index them under the name of the bone anomaly and add the notation "with secondary cavi-

tation of the spinal cord." The small number of cases having both oil and gas myelographies is due to the fact that we have been carrying out both types of myelography on the same patient for only about 1 year. Previous to that time, we did either air or oil myelographies but not both.

TABLE III  
MYELOGRAMS IN SYRINGOMYELIA AND HYDROMYELIA

	Gradation	Proved (20 cases)	Unproved (33 cases)	Disproved (2 cases)
Cord enlarged at oil myelography	Borderline	2	9	1
	Definite	7	5	0
	Total	9/12*	14/23*	1/1*
Cord small at air myelography		6/6*	3/8*	0/0*
Cord small at both air and oil myelography		1/3	0/1	0/0
Cord small at gas but large at oil myelography		2/3	1/1	0/0

\* Remaining patients did not have this study.

## DIFFERENTIAL DIAGNOSIS

The most difficult lesion to differentiate roentgenologically is intramedullary spinal cord tumor. There are certain points in favor of intramedullary tumor such as irregularity of the cord, increased blood vessels on the cord, eccentric position of the intramedullary mass, a mass extending only a few spinal segments and, finally, complete manometric block or complete obstruction to the flow of oil or gas at myelography. Unfortunately, none of these signs is absolute. The presence of congenital bone anomalies, old spinal fractures or osteomyelitis and long standing scoliosis is against spinal cord tumor. High protein content of the cerebrospinal fluid is more frequent in tumors.

In diastematomyelia, an inexperienced observer might conclude that the spinal cord was abnormal in size and/or position. The oil usually runs along one of the two spinal cords or one of the two subarachnoid spaces and superficially might suggest a local expansion of the cord. With repeated attempts in oblique and supine positions, however, the oil can usually be passed along the other spinal cord or the other subarachnoid space, allowing the demonstration of the midline defect characteristic of the diastematomyelia. Of course, in diastematomyelia the bone changes are obviously congenital in nature. The wide spinal canal at the level of the diastematomyelia does not show erosion of the mesial surfaces of the pedicles of the vertebrae and, therefore, does not represent a widened spinal canal.

## CONCLUSIONS

Roentgenograms of the entire spine should be made in all patients suspected of having syringomyelia or hydromyelia. They should be made to search for causes of secondary syringomyelia or to show the presence of a large spinal canal. Oil and also gas

myelography should be done on all such patients. Myelograms should be made in order to find large fluid-filled collections that might be drained with some benefit to the patient and possibly to show an Arnold-Chiari malformation which might suggest that the central canal of the spinal cord is open at the obex. Oil myelography and also gas myelography with the patient in the erect position should be done in an attempt to show a collapsing spinal cord which favors a diagnosis of syringomyelia and/or hydromyelia and would be against a diagnosis of tumor. However, in some patients, it is impossible to differentiate intramedullary spinal cord tumor from syringomyelia by roentgenologic methods.

Oil ventriculography should be considered in some cases in order to discover if the obex is open. It is possible that closing the obex would be beneficial.

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## AN ANATOMIC, MYELOGRAPHIC AND CINEMYELOGRAPHIC STUDY OF THE DENTATE LIGAMENTS\*

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**M**YELOGRAPHIC identification of the dentate ligaments has received little attention. In view of the fact that they can be observed quite often in the cervical spinal canal, and somewhat less so in the thoracic region, a more detailed anatomic and roentgenologic description than is now generally available is of interest. Dynamic changes revealed by cinemyelographic examination are also important in an understanding of the function of the dentate ligaments. The present study is based on dissections of 20 spinal cords removed at necropsy, including 3 examined while the cord was *in situ*. The roentgenologic descriptions are derived from a review of the last 100 consecutive myelograms, all with cinemyelographic records.

### ANATOMY (Fig. 1-4)

The dentate ligaments extend from the midline of either side of the spinal cord as thin longitudinal septa which originate from a barely perceptible thickening of the pia mater. They extend laterally, usually but not always, in a series of 21 triangular folds which insert by means of apical tips into the dura mater. The first dentate segment is situated opposite the margin of the foramen magnum between the vertebral artery and the hypoglossal nerve. The last one is a broader strand of fibrous tissue which unites with its opposite fellow in a fork-shaped extension, merging with the pia mater surrounding the filum terminale.

The dentate ligaments are visible through the translucent intact arachnoid after the dura mater is reflected away. The points of insertion of each triangular fold are extra-arachnoid in position. At the apex of each fold is a small, prong-like con-

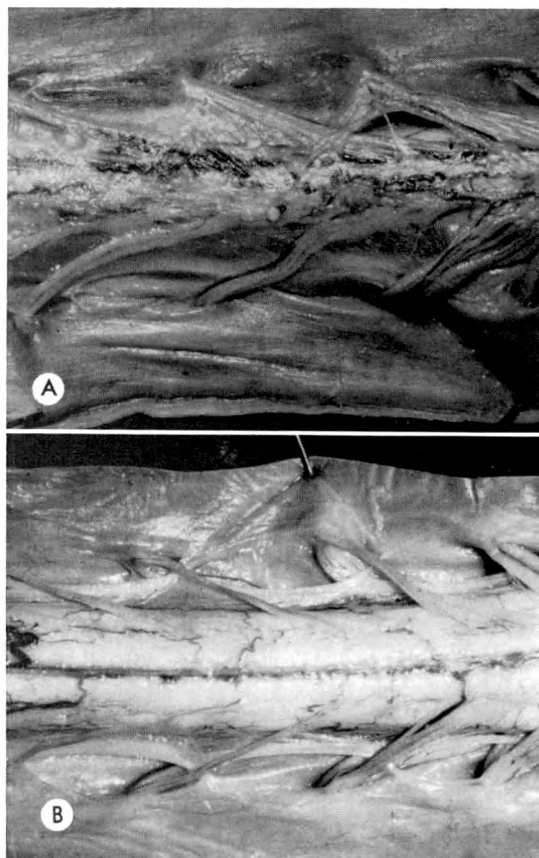


FIG. 1. (A) The dorsal aspect of the dura mater has been incised vertically and reflected away from the intact arachnoid. The cord has been turned slightly so that the left dentate ligament in the midthoracic region is visible to better advantage than the right. (B) The anterior aspect of the dura mater has been incised and reflected laterally. The arachnoid has been removed. Both dentate ligaments extend laterally from the middle of the upper thoracic cord and insert by means of apical tips into the dura mater. The anterior spinal artery and some radicular branches are apparent.

tinuation of fibrous tissue which merges with the dura mater. These prongs are longer in the cervical than in the thoracic

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portions of the cord. In the cervical region, 2 or 3 small continuations of fibrous tissue proceed from the dentate tip to insert into the dura mater. These are about a millimeter in length, and are seen to best advantage when the dura mater is folded back on itself and extended after incision dorsally and ventrally in the midline. In the thoracic region, these extensions usually are replaced by a single heavy fold.

After the arachnoid is stripped away, the dentate ligaments appear as thin sheets made up of delicate interlacing folds of fibrous tissue. Occasional defects are present in the fibrous sheets, some of which probably are artefacts. The triangular leaf-like extensions are smaller in the cervical canal than in the thoracic region, and increase in size caudally. The ligaments are located between the anterior and posterior nerve roots, thereby dividing the spinal canal into an anterior and a posterior compartment. The motor pathways are dorsal to the ligaments and the sensory tracts lie ventrally. Cerebrospinal fluid circulates about the cord, flowing between the compartments through the spaces between the free edges of the dentate ligaments.

In the cervical spinal canal, the dentate insertions are situated about midway between the successive emerging nerve trunks. They are relatively close together because of the short distances between the dural sleeves in this area, and, at rest, are about 2 cm. apart. In the upper thoracic spinal canal, the dural tips are separated more widely, and are furthest apart in the lower thoracic region. The thoracic dural tip insertions are heavier and more blunt than those in the cervical region. They are located more caudally in relationship to the emerging nerve roots than those in the cervical canal. In the lower thoracic region, the points of implantation are close to the caudally placed nerve exits. The fusion of the tips and dura is heavy, and a fold which points cephalad is seen when the dura is stretched. This variation appears to be consistent with the differences in the movement of the spinal cord in the cervical and tho-

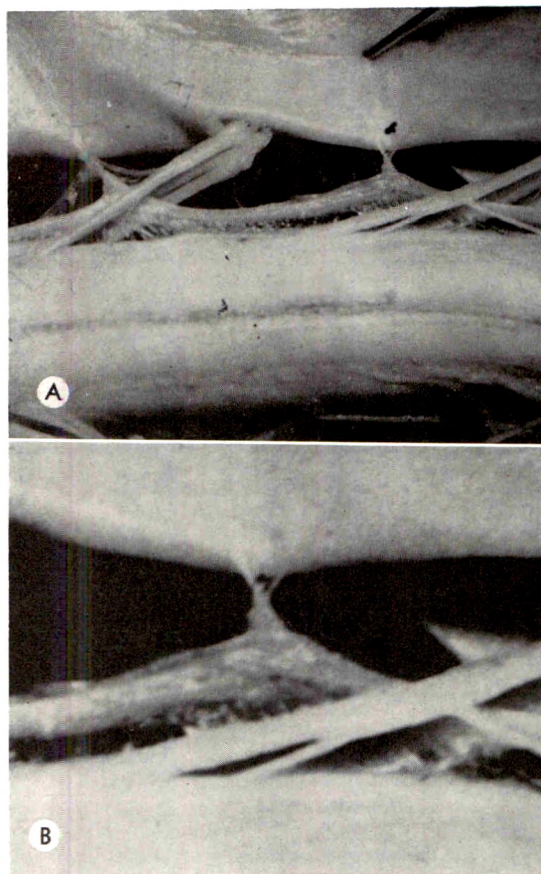


FIG. 2. (A) The dura mater has been incised ventrally and dorsally, and reflected away from the spinal cord. The arachnoid has been removed. When tension is exerted on the reflected dura, the tips of the lower cervical dentate ligament inserting into the dura mater can be demonstrated. Note the rounded, cord-like structure of the lateral margin of the dentate ligament and the filamentous appearance of the triangular extensions. (B) A magnified view of the insertional tip of a cervical dentate ligament reveals a small prong which inserts into the dura mater by means of two small fibrous bands. It is postulated that rotary motion of the dentate ligament takes place by means of this mechanism.

racic regions, a wider range existing in the cervical spine. The last dentate insertion varies in that it is situated some distance from the adjacent emerging nerve trunks. Its insertional point is longer than those immediately cephalad to it.

Of particular interest is the rounded, cord-like structure of the free edge of each dentate ligament. This is a heavy, con-





FIG. 3. (A) The dentate ligament in the lower cervical region is seen through the intact arachnoid. The insertional tips are visible, and join the dura mater about midway between the emerging nerve roots. (B) In the lower thoracic region, the dentate ligament inserts into the dura mater by means of broader tips close to the fusion of the dorsal and ventral nerve roots immediately cephalad. (C) The last dentate ligament is quite large, and its insertional tip is longer than those in the thoracic region. It is some distance from the adjacent caudad and cephalad nerve roots.

←

ligament retracts and the corded lateral margin flattens out into a straight line. When the severed tip is elevated with a fine forceps, the rounded margin returns as the triangular fold is extended.

On manipulating the exposed spinal cord *in situ* and immediately after its removal at necropsy, traction results in a change in the disposition of the dentate ligaments. When the cord is stretched by a directly caudad pull, the dentate ligaments elongate and the distances between the insertional tips increase. This is more prominent in the cervical than in the thoracic spinal canal. The relaxed, cord-like free margins of the dentate ligaments stretch so that the scalloped appearance present at rest is replaced by a more or less straight band, suspended between the successive points of insertion into the dura mater.

On cephalad compression, the dentate tips approximate each other. As the rounded peripheral ligaments relax towards the cord, a more scalloped appearance becomes evident as the triangular folds retract. On lateral flexion, a lengthening of the dentate ligament on the convex side of the cord is seen, while that on the concave side is shortened. The range of movement is greatest in the cervical region. These changes correspond to those described by Breig.<sup>1</sup>

Rotary motion produces a swivelling action of the dentate folds, the rotation centering about the points of insertion of the triangular leaves into the dura mater. In the cervical region, the superior portion of

tinuous, somewhat elastic strand of fibrous tissue, considerably thicker than the lateral triangular sheets or the base of the dentate ligament. The prong-like insertions into the dura mater are extensions of these peripheral ligamentous thickenings. If the dural insertion of a dentate tip is severed, the

the involved segments rotates dorsally and the inferior portion moves ventrally so that the dentate leaves turn obliquely. These changes are exaggerated when observed on manipulation of the cord after removing it from the spinal canal. The range of motion in the thoracic region is limited in this regard.

#### ROENTGENOLOGIC DESCRIPTION AND PROCEDURE

The roentgenologic description of the dentate ligament, at present, is limited to a brief note about their appearance on lateral myelograms.<sup>1</sup> Actual demonstration of their

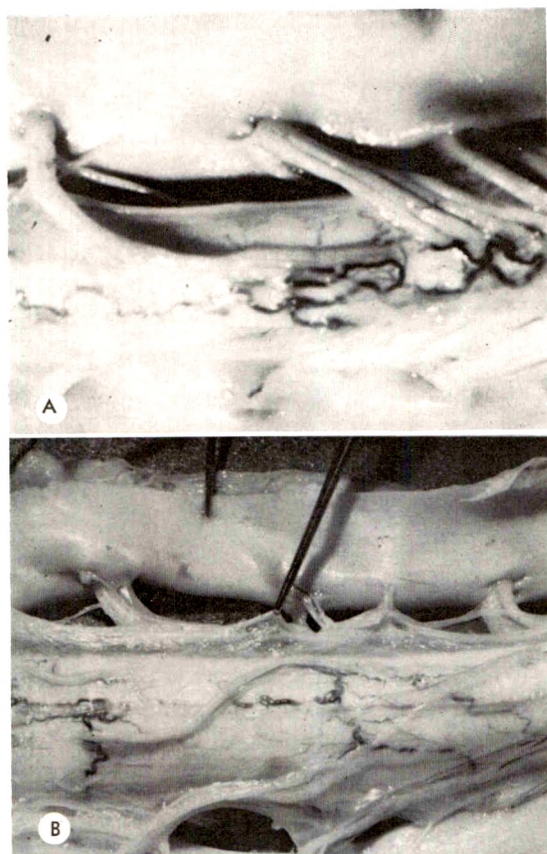


FIG. 4. (A) A dentate tip in the lower cervical region has been severed. The cord-like peripheral band straightens out as the dentate ligament retracts towards the cord. No change was noted in the position of the cord itself. (B) When the severed tip is elevated, the corded peripheral band can again be seen.

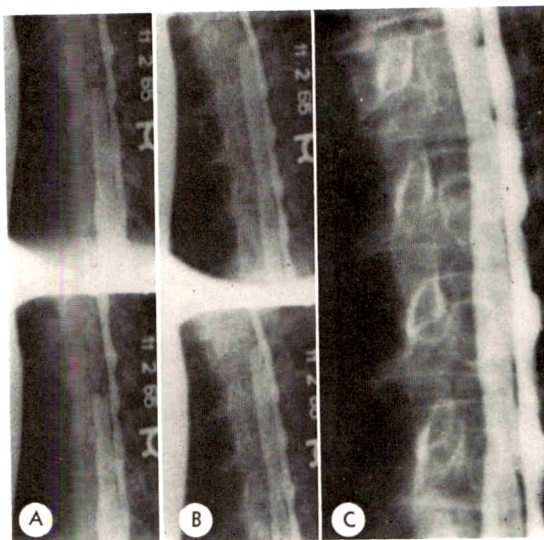


FIG. 5. (A) The patient is turned into an about 60 degree right anterior oblique position. Under image intensification fluoroscopic control, pantopaque flowed under the dependent right dentate ligament more rapidly than above it. These cinemylographic frames show the split pantopaque column. (B) A second or so later, the divided column reunites. (C) A spot myelogram of the same patient. The dependent dentate ligament is visible. The interrupted appearance of the radiolucent line is regarded as representing the triangular leaves.

movement and the different changes in appearance in the various projections used for myelographic examination during life have not been reported. During the past 18 months, this has been studied by means of image intensification fluoroscopy, conventional myelography and cinemylographic examinations (Fig. 5 through 11). While the detail available on spot roentgenograms is far superior to that obtainable from individual cinemylographic frames, examination utilizing both modalities provides a most interesting approach to the study of the dentate ligaments. Movement studies available from repeated viewing of cinemylographic examination have been useful in investigating the alterations in the configurations of the dentate ligaments in flexion, extension and rotation. These observations were correlated with those made at postmortem examinations.

As a rule, about 12 to 18 ml. of pant-



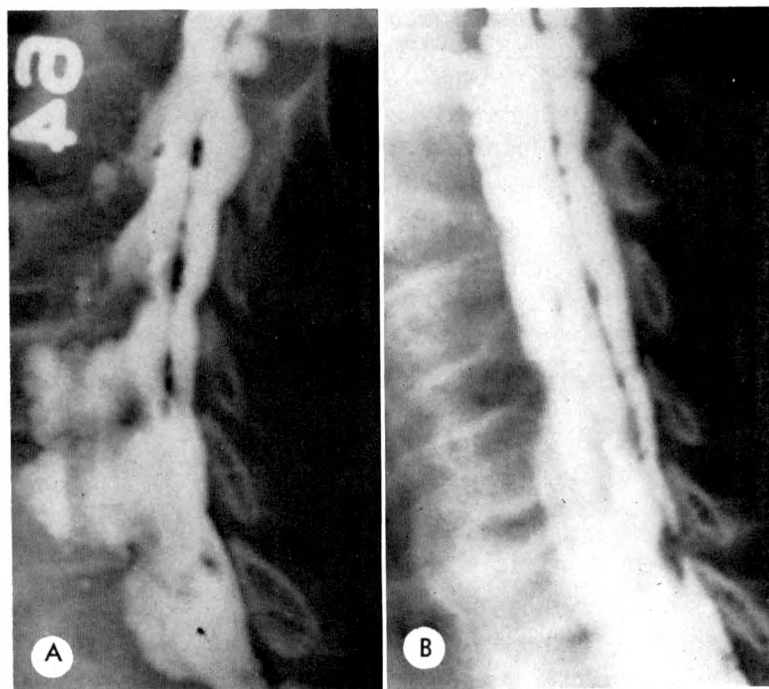
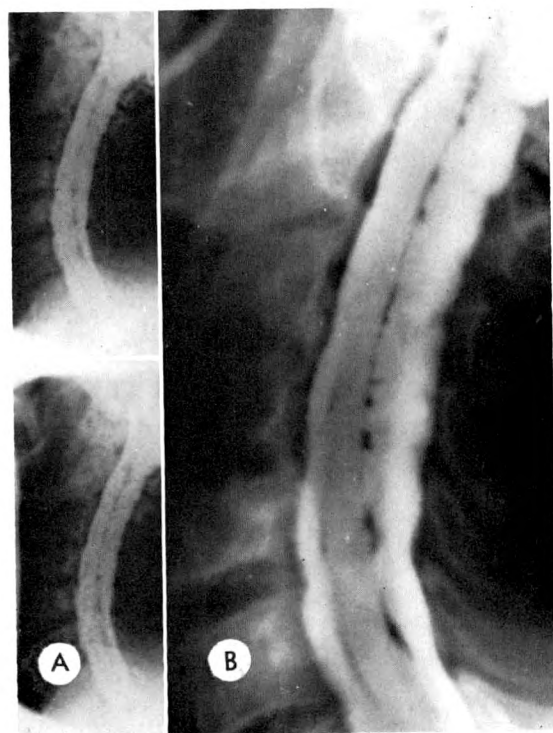


FIG. 6. (A) With the patient in about a 60 degree right oblique position and the head extended, a small quantity of pantopaque has entered the cervical spinal canal. The dependent dentate ligament is apparent. The thicker portions of the broken line represent the tips of the triangular leaves. (B) Note the change in the configuration of the dentate ligament when the neck is flexed. Additional pantopaque has entered the cervical spinal canal during this maneuver.

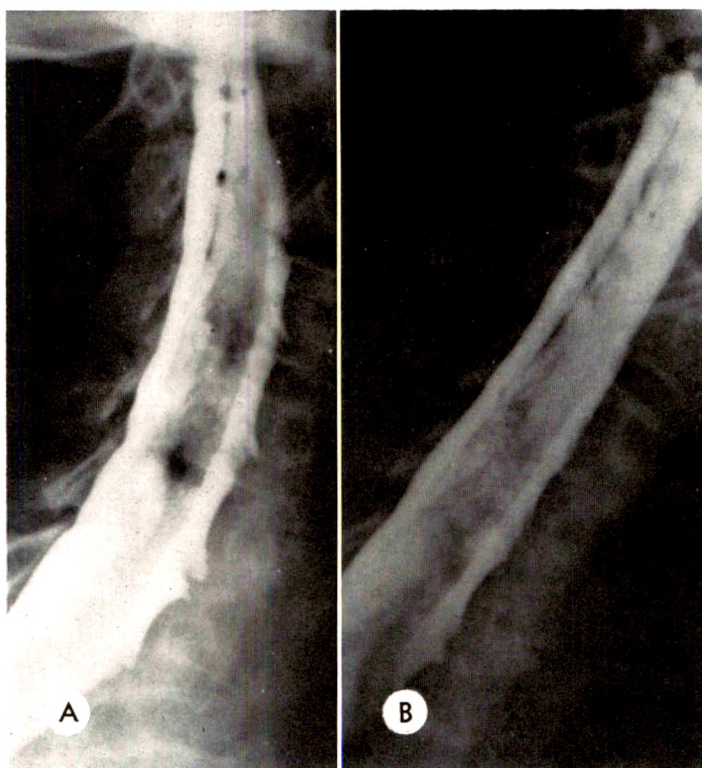


opaque is used for cervicothoracic myelography. The contrast material is introduced from the lumbar sac into the thoracic and cervical spinal canal with the patient turned in about a 45 degree oblique position. The head is postured as for a lateral skull roentgenogram, but is kept elevated. The passage of pantopaque is followed by means of image intensification fluoroscopy. Spot roentgenograms and cineradiographic studies are made as desired. If deemed ad-



FIG. 7. (A) Frames from a cineradiographic run taken with the patient in the recumbent lateral position while extending his neck. (B) Spot myelogram taken at about the same time. The two dentate ligaments are superimposed so that a single midline radiolucent line results. The small rounded shadows are regarded as produced by the tips of the dentate ligaments. The shadow of the cord is visible, and some of the ventral nerve rootlets are seen inferiorly.

FIG. 8. (A) The upper aspect of the cervical dentate ligament is seen on this oblique myelogram made with the neck in approximately a neutral position. The small round radiolucencies represent the dentate insertional tips. (B) The dentate ligament straightens out when the neck is flexed, and the insertional tips become less conspicuous.



visible, pantopaque is permitted to flow into the posterior cranial fossa, taking due precautions to prevent further flow into the middle and anterior cranial compartments.

While traversing the thoracic spinal canal, the head of the contrast column occasionally is divided momentarily by the dentate ligament. With the patient in the prone oblique position, pantopaque passing below the inferiorly placed dentate ligament may proceed for a short distance in advance of the contrast material above the ligament. This results in a split column, with a thin linear asymmetric shadow at the cephalad end of the column which vanishes quickly as the dentate ligament is enveloped in pantopaque. While better seen on cinemylographic records, this phenomenon occasionally can be caught on spot roentgenograms.

The thoracic dentate ligaments appear as fairly uniform radiolucent lines about midway in position as seen in the lateral projection. With the patient obliquely placed, the cephalad aspect is likely to be situated to-

wards the middle of the canal and the caudad portion is inclined dorsally. One ligament is visible when the contrast material fills only the lower half of the spinal subarachnoid space. If the entire canal is opacified, both dentate ligaments are seen occasionally in the oblique projections. In a true lateral projection, they may overlap one another and are represented by a single line. The dentate tips and the apices of the triangular folds appear as interrupted dash-like linear negative shadows. Sometimes, a larger portion of the triangular component of the ligament produces a broader appearance of these segmented shadows.

As pantopaque enters the cervical spinal canal with the patient in the recumbent oblique position, opacification of the dependent surface occurs first. At this time, the inferiorly placed dentate ligament comes into view as a linear radiolucency which may present a discontinuous appearance. With further filling, the ligament sometimes can be identified down to the upper thoracic level. Both dentate ligaments are



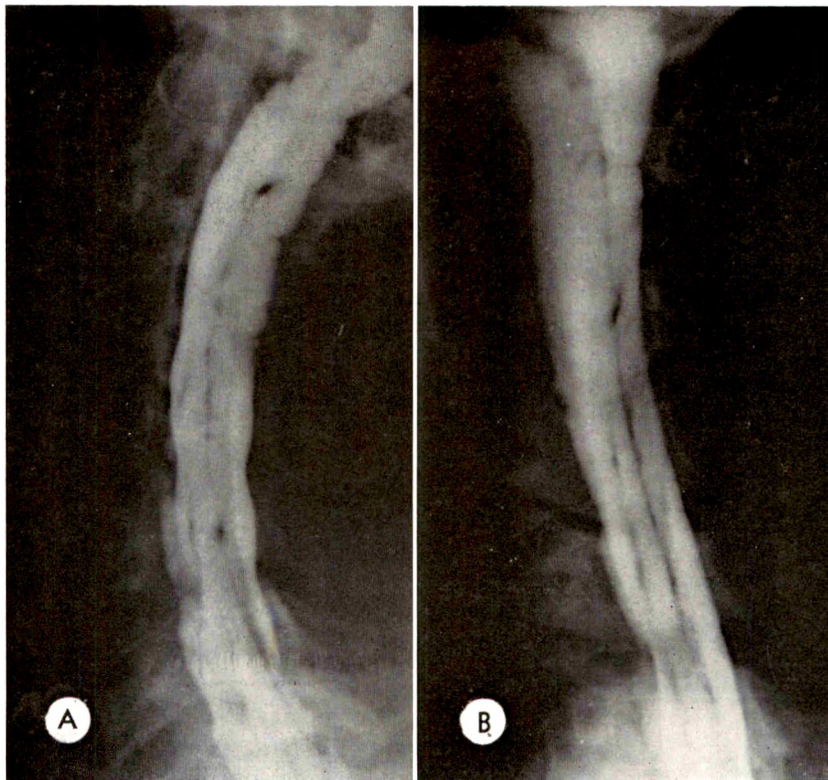


FIG. 9. (A) Cervical myelogram with the patient in the recumbent lateral position and the head moderately extended. Note the oblique position of one of the upper dentate ligament leaves. (B) With flexion and slight rotation, the dentate ligament straightens out and the insertional tips change their position. On this film both dentate ligaments in the lower cervical region can be seen.

identifiable when the canal is filled completely. In lateral projections the dentate ligaments superimpose so that only a single radiolucent line is visible. Small dot-like rounded shadows are seen at intervals. These correspond to the dentate tip insertions into the dura mater. True direct laterals are obtainable in cross-table projections, but cannot be observed fluoroscopically without biplane facilities. It is possible, however, to obtain a similar picture under image intensification control by turning the patient into a lateral position, making sure to control the position of the head to prevent intracranial flooding.

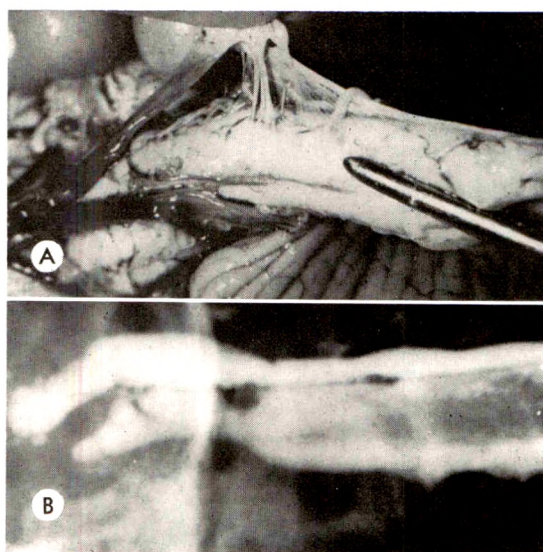
The movement of the dentate ligaments in the upper cervical spinal canal can be observed with only a moderate quantity of pantopaque present. A larger amount is required to fill the canal so that greater lengths of the dentate ligament can be

seen. The first dentate segment can be identified in proximity with the vertebral artery, which is readily demonstrated when the cisterna magna is opacified. A small irregularity appears cephalad, representing the point of origin of the first triangular fold. Proceeding caudad for about a centimeter, a variable ovoid negative shadow appears slightly below the level of the foramen magnum. This is produced by the prominent nerve rootlets of the hypoglossal nerve on either side of the dentate ligament. Rotation of the head results in broadening and shortening of the configuration of this complex.

Motion studies of the dentate ligaments are most striking in the cervical region. When the head is extended, the tips of the dentate folds approximate each other. The rounded points representing the dentate tip dural insertions are about 1.5 cm. apart



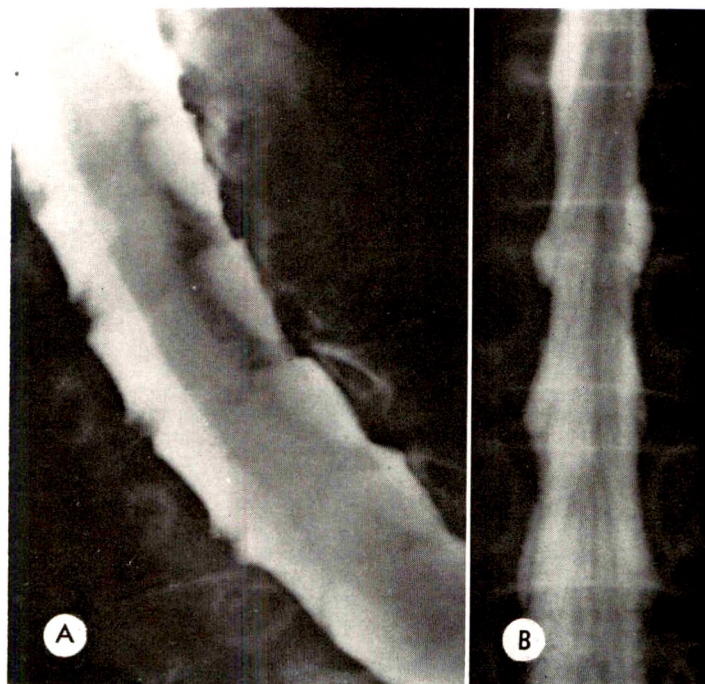
FIG. 10. (A) The first dentate ligament is extended. Note its relationship to the adjacent rootlets of the hypoglossal nerve and the vertebral artery. (B) Lateral cervical myelogram with pantopaque in the posterior cranial fossa. The vertebral artery, the first dentate segment and the defect caused by the hypoglossal nerve can be demonstrated. The insertional tip of the next caudad segment is visible.



when the neck is in the neutral position. They approach each other as the head moves back, and the dentate ligament thereupon assumes a more interrupted appearance. When the neck is flexed, the points of dural insertion become less conspicuous and the distances between them increase to about 2 cm., as compared with the spacing of about 1.5 cm. at rest. The dentate ligament stretches so that the discontinuous configuration of the linear negative shadow of the ligament becomes straightened, continuous and thinner. With rotation of the neck, keeping the head moderately extended, a short oblique shadow appears at some of the dentate tips. The

uppermost point is directed dorsally and the lower margin moves ventrally, much the same as seen in the cadaver when the cord is rotated *in situ*. This represents a swivelling action of the involved triangular sheets of the dentate ligament, the apical point serving as the point about which rotation centers. While these dynamic changes are better observed cinemyelo-

FIG. 11. (A) Oblique cervical myelogram. The rootlets of the spinal nerves extend superiorly as brush-like radiolucencies to emerge from the intervertebral foramina. The lower nerve roots present as trunks, so that the inferior lateral margin of the pantopaque column is broken in appearance. (B) The anterior spinal artery in the lower thoracic spine is seen in the mid-line. Lateral thereto are some of the thoracic nerve roots, passing obliquely to their respective intervertebral foramina.





graphically, they also can be shown on spot myelograms.

#### COMMENT

There is little difficulty in differentiating the dentate ligaments from nerve roots or vascular structures on myelographic examinations. The anterior spinal artery usually is best demonstrated on direct anteroposterior roentgenograms in the midline, and branches passing downwards and laterally towards the axillary pouches in proximity with the nerve roots are quite distinct. This can also be noted on oblique myelograms as well. The nerve roots often are seen as indentations in the lateral aspects of the pantopaque column in the cervical region on anteroposterior myelograms. The rootlets emerging from the cord itself occasionally can be demonstrated as brush-like shadows which converge and continue towards an intervertebral foramen. The anterior spinal artery and some radicular branches appear quite often on thoracic myelograms, but the nerve trunks in this region are rather less prominent. In the region of the conus medullaris, some of the thoracic nerve roots can be demonstrated directed obliquely downwards.

I have not been convinced that any great diagnostic help can be derived from observation of the dentate ligaments. Teng<sup>4</sup> noted that patients with spondylosis or spinal cord tumors may present displacement of the dentate ligaments on lateral myelograms, reflecting the position of these lesions and their displacement of the spinal cord. While this was noted in several of our patients, the other changes associated with intraspinal masses were far more striking.

The question as to whether section of the dentate ligaments is important in the surgical management of cervical spondylosis is open to argument. In the patients seen by

us and operated upon by Dr. Joseph A. Epstein, the dentate ligaments have not been cut. His results have been quite satisfactory.<sup>2,3</sup>

#### SUMMARY

The anatomy of the dentate ligaments is described as seen *in situ* in the cadaver, after removal of the spinal cord at necropsy, and on myelographic and cinemyelographic investigations during life. Prong-like protrusions are present at the apical tips of each triangular leaflet. These emerge from a cord-like rounded peripheral band which circumscribes the free edge of each dentate ligament, and inserts into the dura mater. Variations in the cervical and thoracic regions are noted. These dural insertions function as points around which the segments of the dentate ligament swivel or turn during rotary motion.

These changes, as well as those apparent with flexion and extension, were studied in the cadaver and during life. Cinemyelographic examinations were found to be desirable for motion studies.

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## TRAUMATIC EXTRADURAL CYSTS OF THE SPINE

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**I**N A review of the literature, scant reference has been made to traumatic extradural cysts of the spine. We wish to report 2 cases of traumatic extradural cysts in which the dura was inadvertently opened during lumbar disk surgery. In neither case was there any evidence of previous infection present when the traumatic cyst was discovered. A review of the literature is also presented.

Although the dura is frequently traumatized during lumbar punctures and surgery, it is surprising that only 11 cases of extradural cysts resulting from disk surgery have been reported in the literature.<sup>1,12,19,22,24,27,28,30</sup> Three further cases are referred to by Lombardi and Passerini.<sup>13</sup>

This condition may be relatively asymptomatic or perhaps patients may accept some degree of discomfort in the presence of a cyst without subjecting themselves to re-examination. Another explanation for so few reports may be the fact that post-operative myelography is not performed routinely as a long term follow-up after disk surgery.

### REPORT OF CASES

**CASE 1.** V. McC. (S.V.H. & M.C., No. 119470) (Fig. 1 through 3). A 60 year old obese female was admitted to the hospital on July 13, 1960 with pain in both lower extremities, more marked on the left side. There was no history of injury although this patient had always done rather heavy work. The pain had come on suddenly, was sharp and constant in nature and radiated from the buttocks to the medial and lateral aspects of the left thigh and knee.

Physical examination revealed limitation of movements of the left leg and thigh due to pain. The left knee jerk was more brisk than the right. Bilateral plantar responses were present. A sensory examination revealed hyperesthesia of the upper two-thirds of the anterior aspects

of the left thigh, extending laterally and medially.

Plain roentgenograms of the spine demonstrated no abnormality. A pantopaque myelogram showed changes consistent with marked disk protrusion at the level of L3-L4, probably midline.

A laminectomy shortly thereafter showed a large protruded disk at the L3-L4 interspace which was removed. The patient had an uneventful recovery and was symptom free when discharged from the hospital.

Three-and-one half years later, the patient was re-admitted, complaining of a sudden onset of pain in her back related to a bout of coughing 3 weeks prior to admission. There was intense, constant low back pain, more pronounced over the right sacroiliac joint, not relieved by rest and medications. On examination, there was severe paravertebral muscle spasm with marked tenderness over the scar of the previous operation. Jugular vein compression increased the pain slightly. The right knee jerk was diminished.

**Myelographic Findings.** In an attempt to puncture the lumbar subarachnoid space, the needle immediately entered a cavity from which there was a free flow of clear cerebrospinal fluid. During passage of the needle through the tissues, the normal resistance of ligaments and dura appeared to be diminished. Nine cc. of pantopaque was introduced and fluoroscopy revealed a large extradural cavity which communicated freely with the spinal subarachnoid space. The cyst was located at the level of L4-L5. The pantopaque was not removed at this stage and the patient was re-screened 24 hours later. The cyst was again outlined and communicated freely with the subarachnoid space.

**Operative Findings.** The patient was re-explored on February 21, 1964. After incising the skin, a cystic bulge was encountered. This was opened longitudinally and a cavity with a smooth lining was exposed. The cavity measured 2X4 cm. and contained cerebrospinal

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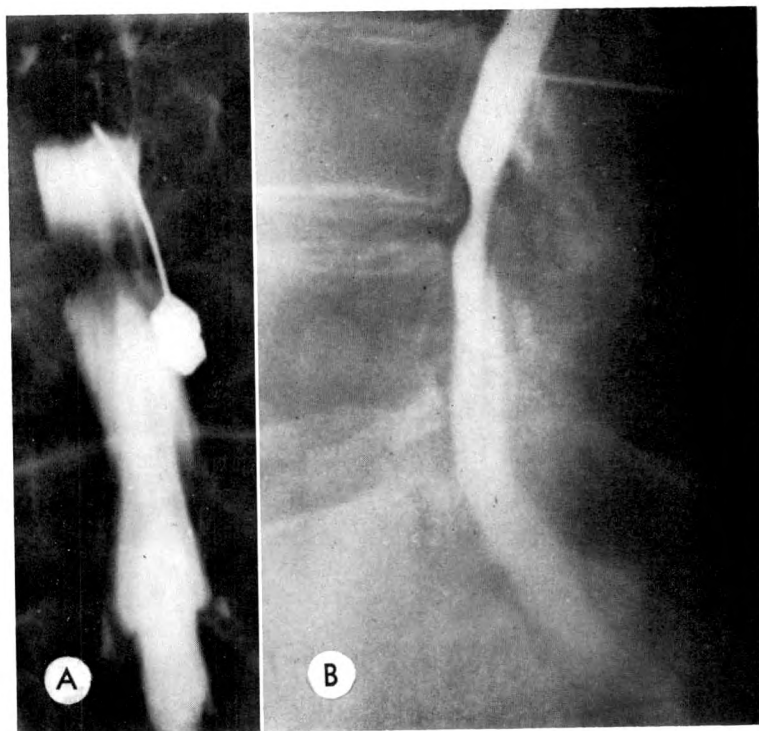


FIG. 1. Case I. (A and B) July 14, 1960: Myelograms showing disk herniation at L3-L4 level, which was removed.

fluid. With further dissection, a tiny communicating channel leading to the subarachnoid space was discovered. A plastic repair was carried out by swinging a pedicle of muscle into the cavity to seal this defect in the dura and the cavity was then closed. Recovery was uneventful and the patient remained asymptomatic.

CASE II. C.T. (B.G.S.M. No. 009070) (Fig. 4, A and B). This 60 year old physician was seen on February 6, 1957 with a history of having had his first attack of low back pain at the age of 20, when he lifted some heavy construction material. The attack lasted 2 days. A few similar attacks occurred over a period of years and at the age of 53, the pain became more constant in his low back with radiation into his left hip and down his left leg. He was admitted to the hospital 2 years later and a laminectomy with removal of a protruded disk at the level of L5 was performed. The patient recovered and had little difficulty, but returned 11 years later with pain radiating down the lateral aspect of his left leg into the left foot, with the clinical impression of a recurrent herniated nucleus pulposus at the L5-S1 interspace on the left.

*Myelographic Findings.* Myelography revealed a defect at the L4-L5 interspace on the left side and a pseudocyst or meningocele at the L5-S1 level on the left.

*Operative Findings* (February 8, 1957). About 1 cm. deep to the level of the spinous processes, a cystic structure was encountered which was eventually found to communicate directly with the subarachnoid space at the level of L5 where a small 2 mm. opening was discovered in the dura. The cyst measured about 3×6.5 cm. in diameter. The opening in the dura was closed with silk sutures. The L4-L5 level was then explored and a disk extrusion at this level was removed.

#### DISCUSSION

In a review of the literature, numerous reports of congenital extradural cysts were found. These were first clinically documented by Elsberg, Dyke and Brewer<sup>5</sup> in 1934. The roentgenologic changes include widening of the spinal canal, erosion of pedicles, irregularity of epiphyseal plates (epiphysitis), and a widening of the para-

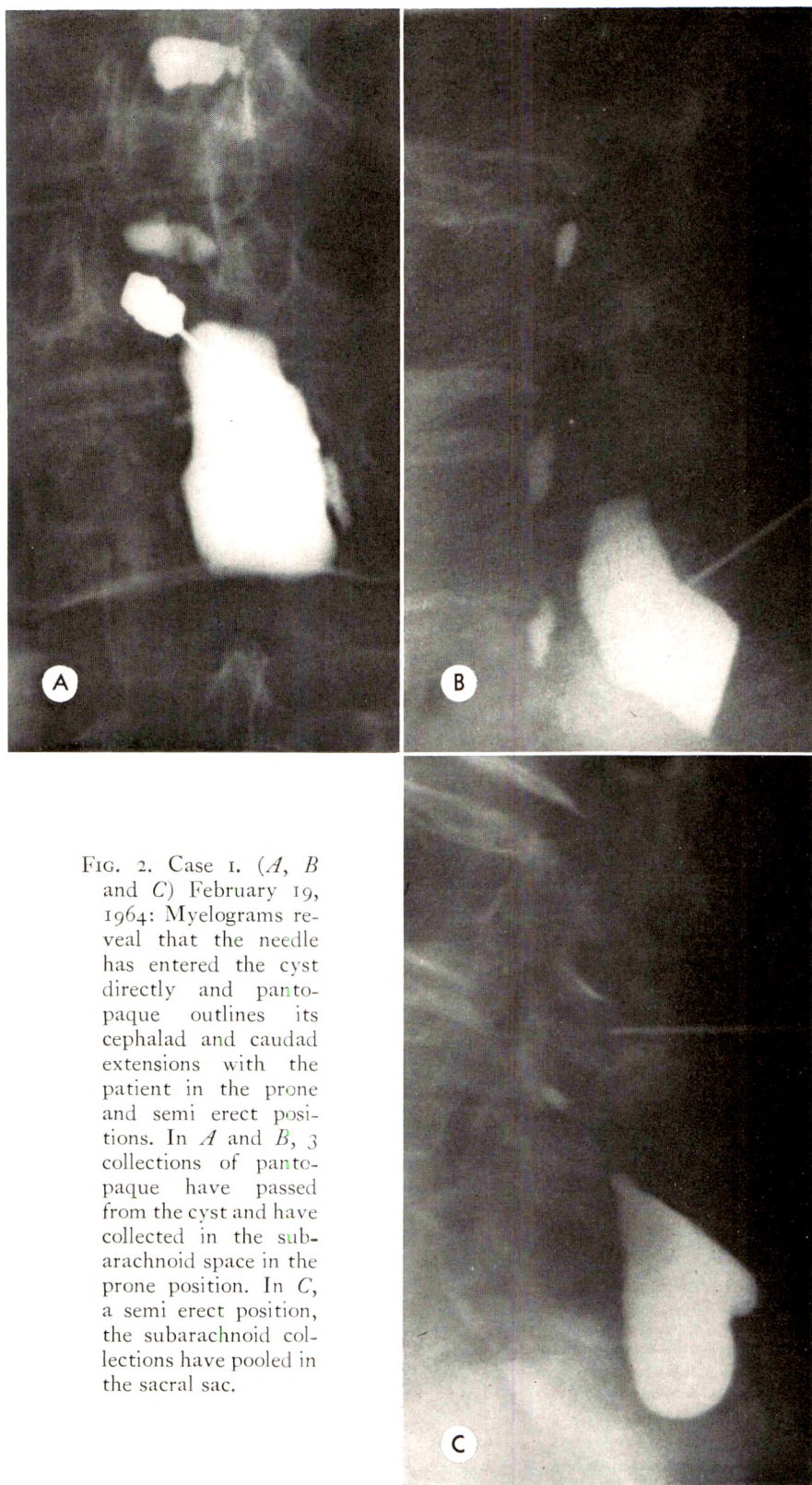


FIG. 2. Case 1. (*A*, *B* and *C*) February 19, 1964: Myelograms reveal that the needle has entered the cyst directly and panto-paque outlines its cephalad and caudad extensions with the patient in the prone and semi erect positions. In *A* and *B*, 3 collections of panto-paque have passed from the cyst and have collected in the sub-arachnoid space in the prone position. In *C*, a semi erect position, the subarachnoid collections have pooled in the sacral sac.



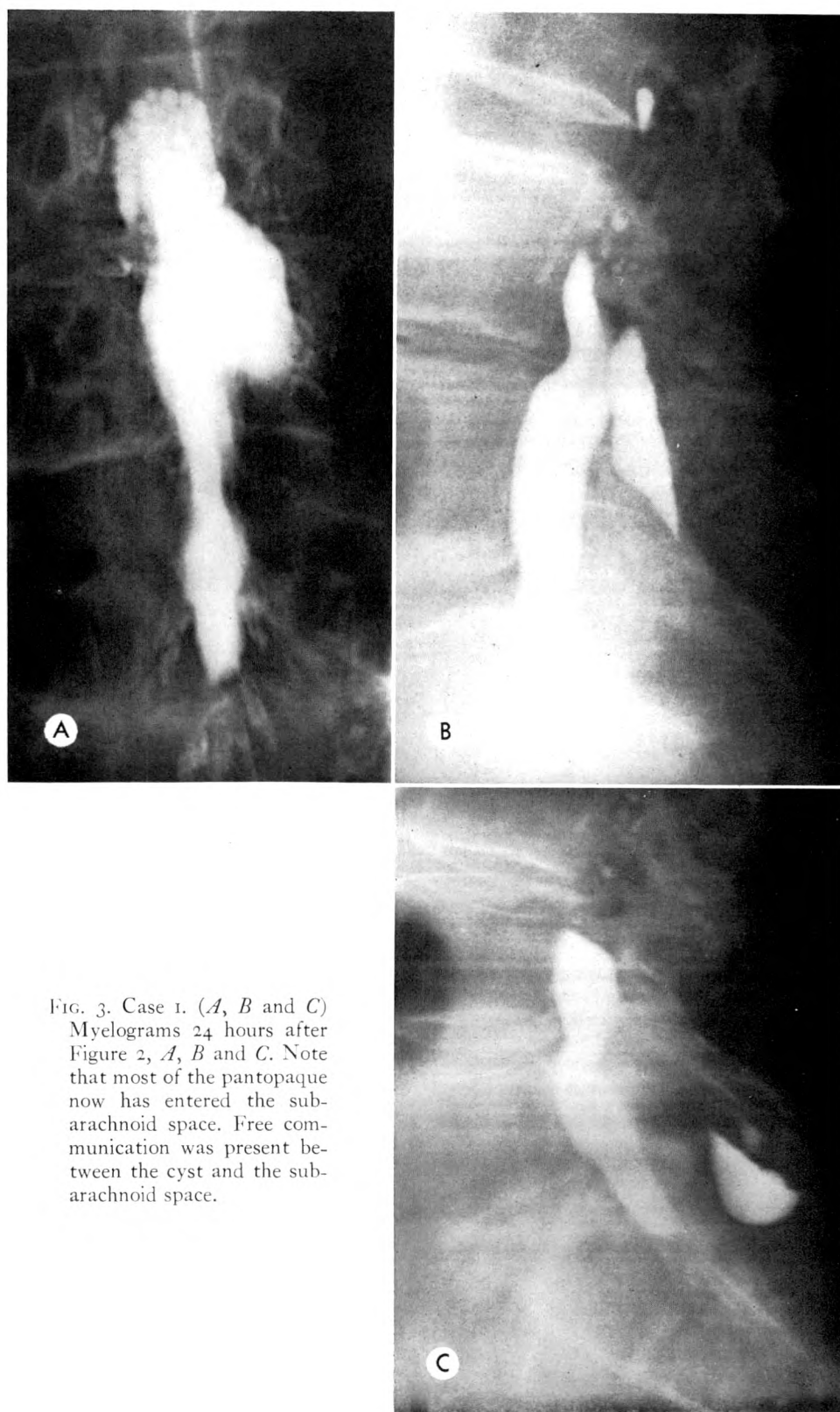


FIG. 3. Case 1. (*A*, *B* and *C*) Myelograms 24 hours after Figure 2, *A*, *B* and *C*. Note that most of the pantopaque now has entered the subarachnoid space. Free communication was present between the cyst and the subarachnoid space.

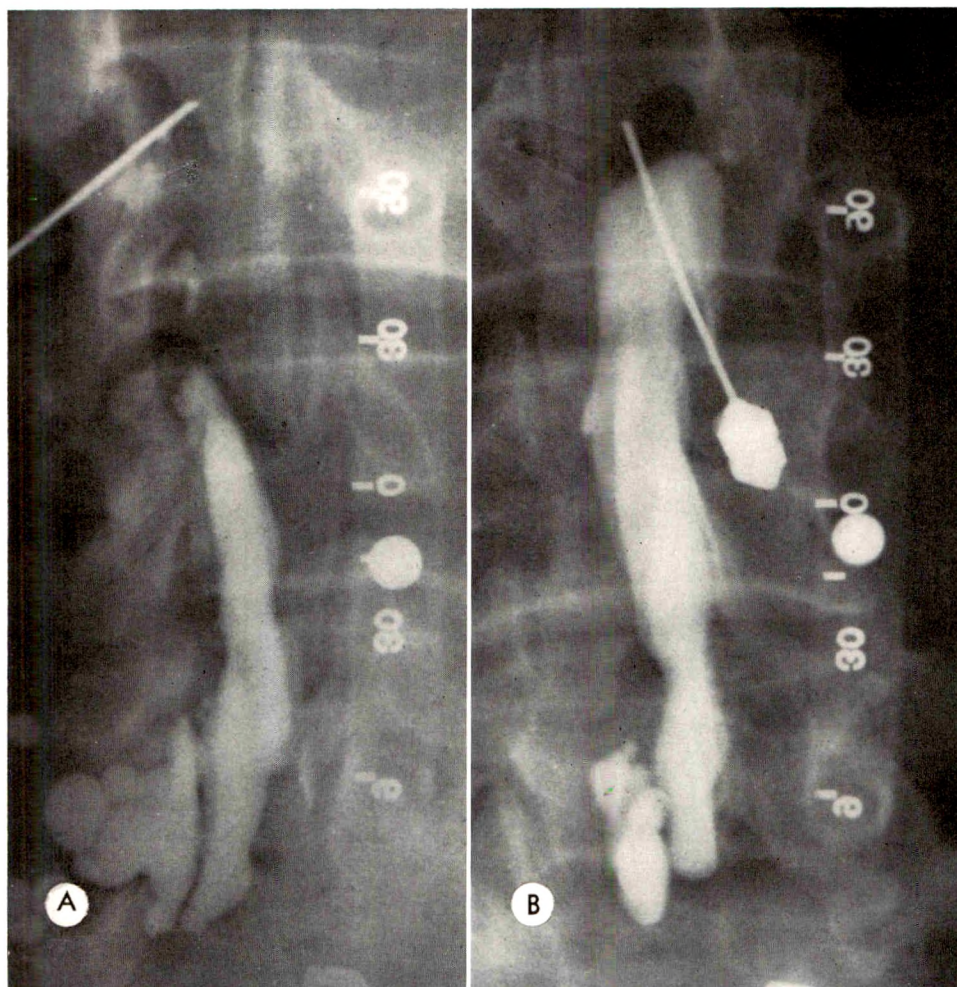


FIG. 4. Case II. (A and B) Myelograms made 11 years after the removal of a disk at the level of L5. Pantopaque passed freely from the subarachnoid space to the cyst via a communication. A defect in the pantopaque column at the level of L4-L5 is noted. At surgery a herniated disk at this level and the cyst at the level of L5-S1 were removed.

vertebral shadows.<sup>3,5,8,11</sup> Myelograms in the majority of cases show a partial or complete block to the passage of pantopaque in the subarachnoid space. A communication between the cyst and the subarachnoid space is rare.<sup>11</sup>

We will limit our discussion to extradural cysts of traumatic origin.

**Etiology.** In some cases, a congenital weakness in the dura prior to trauma may predispose to cyst formation. A defect in the dura allows the arachnoid to herniate and perpetuate the cyst formation.<sup>15,17,27,28</sup> This is probably the mechanism in most

cases of closed injury to the spine. Open injuries of the dura may result from multiple needle punctures,<sup>6,9</sup> surgical exploration of spinal compartments deep to the dura<sup>10</sup> and inadvertent trauma to the dura during thoracoplasty.<sup>14,20</sup> The dura may also be torn by injuries involving the spinal column.<sup>2,7,18,26</sup>

When the dura is opened, the cerebrospinal fluid may escape into the extradural tissues. This fluid is usually absorbed during the initial stages following trauma. If the opening in the dura remains patent, the absorption mechanism of the escaping



cerebrospinal fluid will be deficient as the fluid sets up a connective tissue reaction and a nonabsorbing membrane will gradually develop.<sup>10,19</sup>

*Age Incidence.* Although most disk surgery is undertaken during adult life, cases of extradural traumatic cysts have been reported in infants following injury to the spine.<sup>28</sup>

*Sex.* Traumatic extradural cysts of the spine are found predominantly in males because of occupational hazards.

*Location.* Most occur in the lumbar region, although cysts have appeared in the cervical, thoracic and sacral regions.<sup>4,10,16,17,21,22</sup>

*Time Interval.* The interval between trauma and discovery of cyst varied from a few months<sup>10,16,30</sup> to many years.<sup>1,22,27,28</sup>

*Symptoms.* When related to disk surgery, the symptoms were usually a recurrence of those present before surgery. These symptoms and signs were mainly related to pressure on nerve roots by the cyst or herniation of nerve roots into the cyst.<sup>12,27</sup> Since the canal in the sacral region is relatively capacious, the onset of pressure symptoms may be delayed when compared with lesions in the cervical and thoracic region.<sup>22</sup> In most cases, no cyst was clinically evident; in a few instances, however, the cystic mass was apparent.<sup>23,28</sup>

*Histology of Cyst Wall.* This is usually dense fibrous tissue lined by mesothelial-like cells.<sup>10,26</sup> If the arachnoid herniates, the cyst wall will be lined by this membrane.<sup>25</sup>

*Roentgenographic Findings.* Plain roentgenograms of the spine are usually unrevealing. Evidence of previous surgery or trauma may be present. No reports have appeared of cases showing changes in the spinal pedicles or canal as is usually seen associated with the congenital type. Two cases revealed evidence of ossification of the cyst wall.<sup>22,29</sup>

*Myelographic Findings.* In our first case report, the needle entered directly into the cyst cavity. In retrospective reasoning, this also occurred in a case reported by

Swanson and Fincher.<sup>28</sup> Other reports of this occurring have appeared with only the congenital cyst variety. In all cases of traumatic extradural cysts, there was free communication between the subarachnoid space and the cyst cavity. The communicating tract was not always demonstrated.

In the evaluation of patients who have recurrence of symptoms following disk surgery, extradural cyst formation, although rare, must be kept in mind and a thorough roentgenologic examination with particular emphasis on myelography is essential. This should include fluoroscopy in the prone, supine and lateral decubitus positions. Myelography in the prone position alone may be unrevealing since the cysts are usually located on the dorsal aspect of the subarachnoid space. Delayed screening should be undertaken, since pantopaque may not immediately find its way into the cyst.

Occasionally, the cyst will cause a complete or incomplete block to the passage of pantopaque in the subarachnoid space. Such a block may be misinterpreted as recurrence of tumor.<sup>10</sup>

#### SUMMARY

Two cases of traumatic extradural cysts of the spine are reported following disk surgery. Both cases had complete myelographic studies and the correct diagnosis was made prior to surgery.

The rarity of this entity is evidenced by only 11 documented case reports in the literature.

Careful myelographic examination is stressed in the evaluation of patients with recurrent symptoms following disk surgery.

The literature has been reviewed and discussed.

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# VENOUS INTRAVASATION OF IODIZED OIL DURING MYELOGRAPHY\*

## REPORT OF A CASE WITH ROENTGENOLOGIC EVIDENCE OF CONTRAST MEDIUM IN THE ILIAC VEINS AND IN THE LUNGS

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WHEN performing myelography, we have noticed in 9 patients a sudden disappearance of the iodized oil from the spinal subarachnoid space during the phase of its removal following completion of the procedure. Eight of these cases have been reported previously.<sup>2</sup> The hypothesis was advanced that the opaque medium might have entered the venous system following accidental puncture of the vertebral plexus. Actually, bloody spinal fluid was obtained through the needle in every one of these 8 cases during the aspiration of the oil. At that time, however, no roentgenologic evidence of contrast medium in the venous system could be obtained.

More recently, we have observed the unequivocal roentgenologic evidence of such an occurrence in an additional case, with the presence of the contrast material in the sacroiliac veins and in the lungs.

### REPORT OF A CASE

The patient had been admitted with a history of progressive tetraparesis of some years' duration, more marked on the right side, and with a sensory loss level at C<sub>4</sub>. Lumbar puncture showed normal manometric and cytochemical values, with the exception of the benzoin curve (0000.0222.0000.0000).

Roentgenograms of the spine showed no pathologic findings. Myelography was then performed with the introduction of 9 cc. of myodil. Clear colorless cerebrospinal fluid was obtained on lumbar puncture. The contrast medium was followed under fluoroscopy from the cul-de-sac to the foramen magnum and no abnormalities could be observed (Fig. 1). At the end of the procedure, a second spinal puncture

was made between L<sub>5</sub> and S<sub>1</sub>, with the patient in the sitting position, in order to remove the contrast material; bloody spinal fluid, but no oil was obtained. At this stage, the patient had some bouts of dry cough and presented for a few minutes mild signs of circulatory collapse, with skin pallor, hypotension, etc. A roentgenogram of the lumbosacral segment (Fig. 2) showed the oil to have almost disappeared from the sac, while the sacroiliac veins were opacified.

Two minutes later, a chest roentgenogram revealed the presence of thin opaque streaks in both pulmonary bases (Fig. 3). Fifteen minutes later, a control roentgenogram of the chest demonstrated their complete disappearance.

The patient experienced no further discomfort and was transferred some days later to a neurological service, with the diagnosis of multiple sclerosis of the cord.

### DISCUSSION

Ten similar cases have been reported in the literature.<sup>1,3-10</sup> Only in 5 of these was there roentgenologic evidence of contrast medium in the lungs obtained; in the other 5 patients, the positive findings were limited to the sacroiliac veins. This complication occurred during myelography, either at the moment of lumbar puncture or during movements of the patient with the needle *in situ*.

In our own cases,<sup>2</sup> it always occurred at the end of the procedure during the extraction of the opaque medium. Roentgen examination of the chest was performed, however, only in the patient reported here.

It is possible that this accident has been met with a relatively higher frequency in our series of myelographic studies because

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of the repetition of the lumbar puncture that we customarily do in order to allow exploration of the dorsal as well as of the ventral surface of the spinal canal. Obviously, repetition doubles the chances of puncturing the vertebral venous plexus.

This uncommon accident during myelography deserves some consideration if we remember Strain's<sup>9</sup> experimental observations. While rapid intravenous injection of 8 cc. of pantopaque in dogs could be fatal, slow injection of the same amount of contrast medium produced no ill effects.

In our 9 cases, no untoward effects were noted with the exception of a slight, transi-

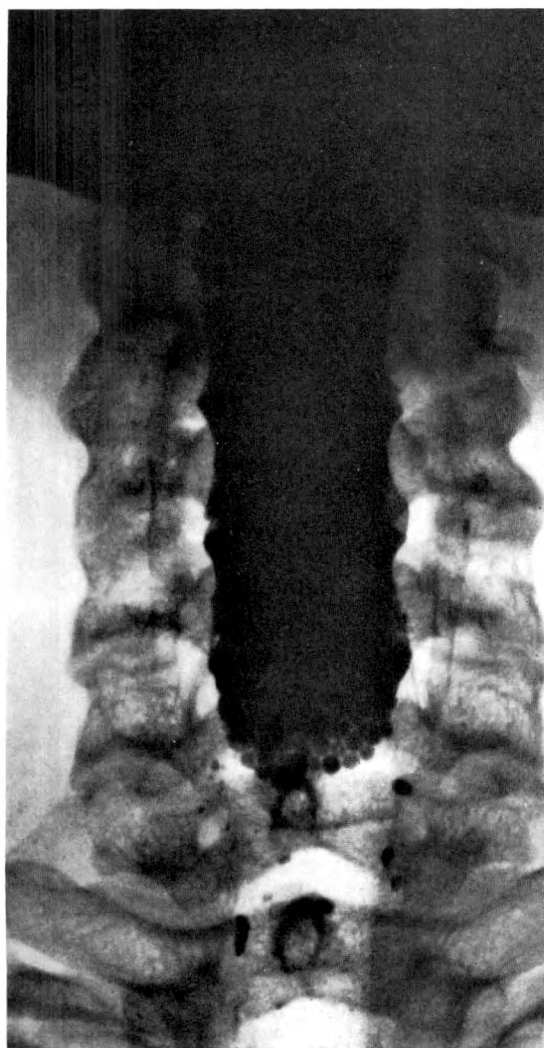


FIG. 1. Myelogram of the cervical tract showing normal findings.



FIG. 2. Myodil in the sacroiliac veins.

tory circulatory collapse and a dry cough.

Schultz and Miller<sup>6</sup> have proposed the injection of only a small quantity of contrast medium into the spinal canal at the beginning of myelography if bloody cere-



FIG. 3. Opaque streaks in the right pulmonary base.



brospinal fluid is obtained. In our opinion, it might be wise in this event to postpone the procedure altogether.

#### SUMMARY

The entrance of iodized oil into the venous system represents a possible, although rare, complication of myelography, which usually does not result in any permanent clinical consequences.

A case of intravasation with the roentgenographic demonstration of contrast material in the sacroiliac veins and in the lungs is reported.

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## ANGIOGRAPHIC FINDINGS IN A PATIENT WITH TUBEROUS SCLEROSIS\*

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A PATIENT with the typical clinical and roentgenographic abnormalities of tuberous sclerosis was thought to have "polycystic disease of the kidneys" at 2 previous institutions on the basis of intravenous pyelograms. This patient was studied by selective renal and celiac angiography, which instead disclosed hamartomas of the kidneys and liver. This case is described and illustrated in order to stress the importance of angiography in the evaluation of patients with unilateral or bilateral nephromegaly.

### REPORT OF A CASE

A. C., a 25 year old white woman, was first admitted to Jackson Memorial Hospital, Miami, Florida, in October, 1964 with 2 days of chills, fever and constant dull nonradiating right flank pain. The patient has been afflicted with epilepsy since the age of 3 years which is controlled by medicine. There is no family history of any kind of kidney disease, abdominal masses, hypertension, skin lesions, mental deficiency, or epilepsy. The present illness began 5 years prior to admission when she noticed bilateral abdominal masses which slowly but progressively enlarged. In 1961 after intravenous pyelography was performed, she was explored through a right flank incision at a hospital in Ohio, and was told that she had "cystic disease" of her kidneys. The patient had had 2 normal pregnancies and delivered in 1962 and 1964. The masses continued to grow slowly except during each pregnancy when there was definitely accelerated enlargement which did not regress after delivery. An intravenous urogram in 1962, after her first pregnancy, was again diagnosed as "cystic kidneys."

For several months prior to the current admission, she had experienced increasing polyuria, and recently had developed dysuria. There

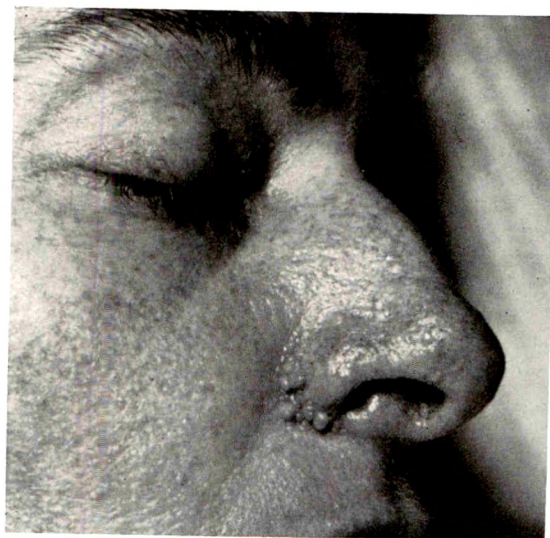


FIG. 1. Lateral photograph of the face showing several angiofibromas (so-called "adenoma sebaceum") in the nasolabial fold.

is no record of any hypertension, hematuria, or previous urinary tract infections.

Physical examination showed a chronically ill, pale, slender, white woman with a blood pressure of 120/65. Pertinent physical findings included large, asymmetric, protruding, lobular masses in each upper quadrant of the abdomen which were slightly movable and ballotable, with the right side larger than the left. The mass borders were poorly demarcated but extended into the pelvic cavity, while the medial aspect projected to the midline. No other organomegaly was found. Neurologic examination was within normal limits. There were several firm shiny nodules of small but variable size in the nasolabial area of the face (Fig. 1). Subungual warts were present on several fingers (Fig. 2) along with café-au-lait spots on her thighs, and shagreen plaques on her back. Funduscopic examination disclosed yellowish retinal plaques (Fig. 3).

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FIG. 4. (A) Intravenous and (B) retrograde urographic studies demonstrating deformed, dilated collecting systems, secondary changes produced by intrarenal kidney masses, ill-defined kidney outlines, and medial displacement of the proximal right ureter. Spotty areas of osteosclerosis are present in both iliac bones. These areas simulate osteopoikilosis, bone islands, and even osteoblastic metastasis.

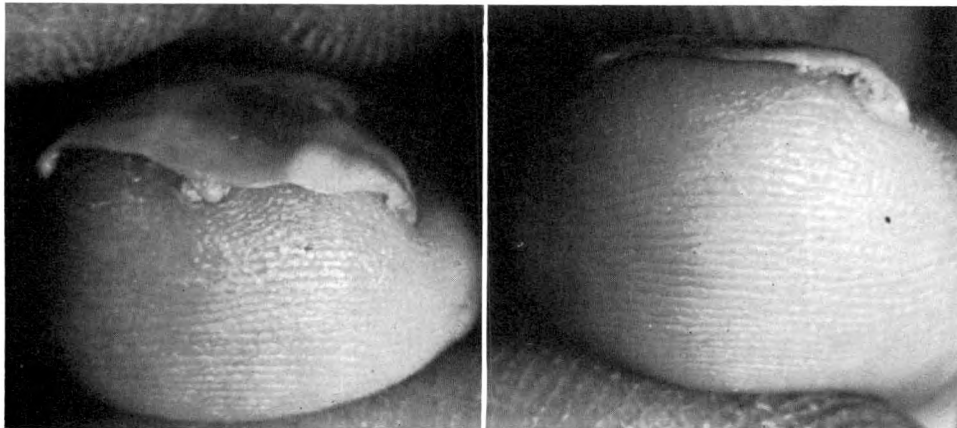


FIG. 2. Close-up photographs of the tip of the fingers showing subungual fibromas (believed to represent a pathognomonic finding in tuberous sclerosis) and deformity of the finger nails.

Pertinent laboratory findings included the following: hematocrit 24 per cent with a normocytic, normochromic red blood cell morphology; and a white blood cell count of 15,600 with a shift to the left. Urinalysis disclosed specific gravity of 1.012, 50 mg. per cent of protein, negative sugar, and many bacteria and white

blood cells microscopically. Urine culture grew out *Proteus vulgaris*. Blood urea nitrogen was 34 mg. per cent; serum creatinine 1.2 mg. per cent; and phenolsulfonphthalein excretion was 19.3 per cent in 2 hours. Liver function studies included bromsulphalein of less than 5 per cent retention in 45 minutes; alkaline phosphatase of 2.1 Bodansky units; and total protein of 6.5 gm. per cent with A/G ratio of 2.99/3.6. Electrocardiogram and electroencephalogram were both within normal limits.

Intravenous urography and retrograde pyelography disclosed bilaterally enlarged kidneys with compressed, dilated calyces and distorted renal pelves. There was superior and medial displacement of the proximal third of the right ureter, which was extrinsically compressed by the enlarged lower medial aspect of the right kidney (Fig. 4, A and B). Cystoscopic examination was unremarkable except for trigonitis.

Selective renal angiograms (Fig. 5, A and B; and 6, A and B) demonstrated marked hypervascularity with angiomatous malformations in both kidneys. There were, however, no signs of arteriovenous fistulae. The hypervascularity had an asymmetric distribution and the posterior two-thirds of the right kidney appeared avascular (Fig. 6, A and B). Selective celiac

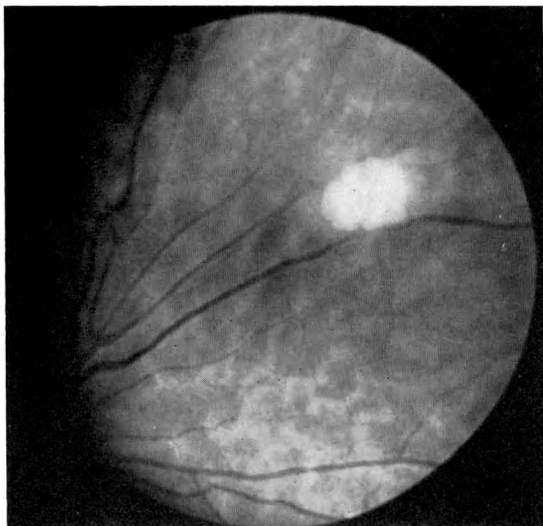


FIG. 3. Fundusoscopic photograph showing a large yellowish plaque, so-called phacoma of the retina.

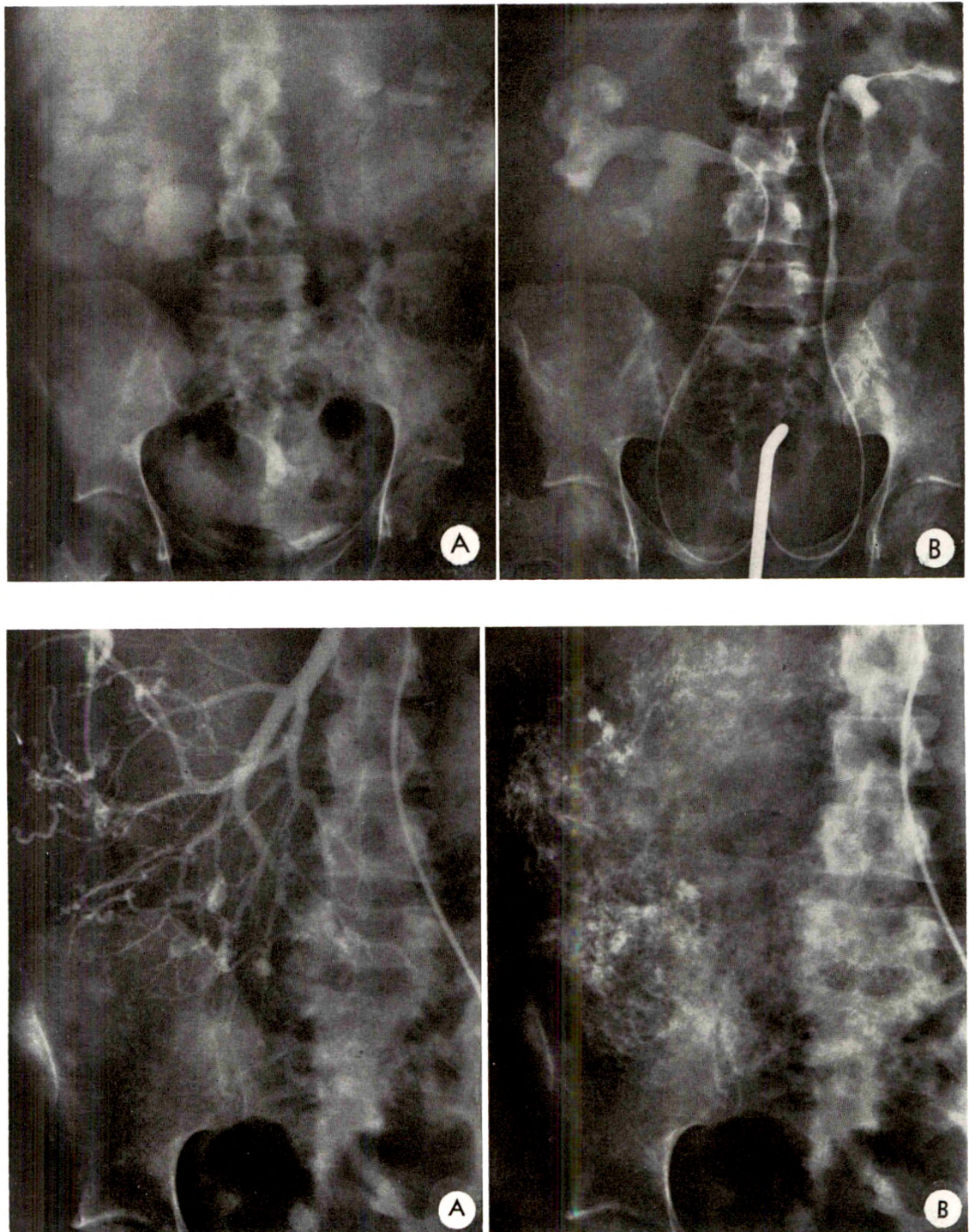


FIG. 5. Selective right renal arteriograms in the arterial (*A*) and nephroangiographic (*B*) phases. The main renal artery and interlobar branches are large and show variable degrees of displacement. The contrast material pools and remains temporarily in small berry-like vascular collections in direct continuity with interlobar and interlobular branches of the renal artery. The collection of contrast material is in arterial angiomatous malformations. There is no evidence of early venous phase. The lack of early appearance of the veins rules out arteriovenous fistula.



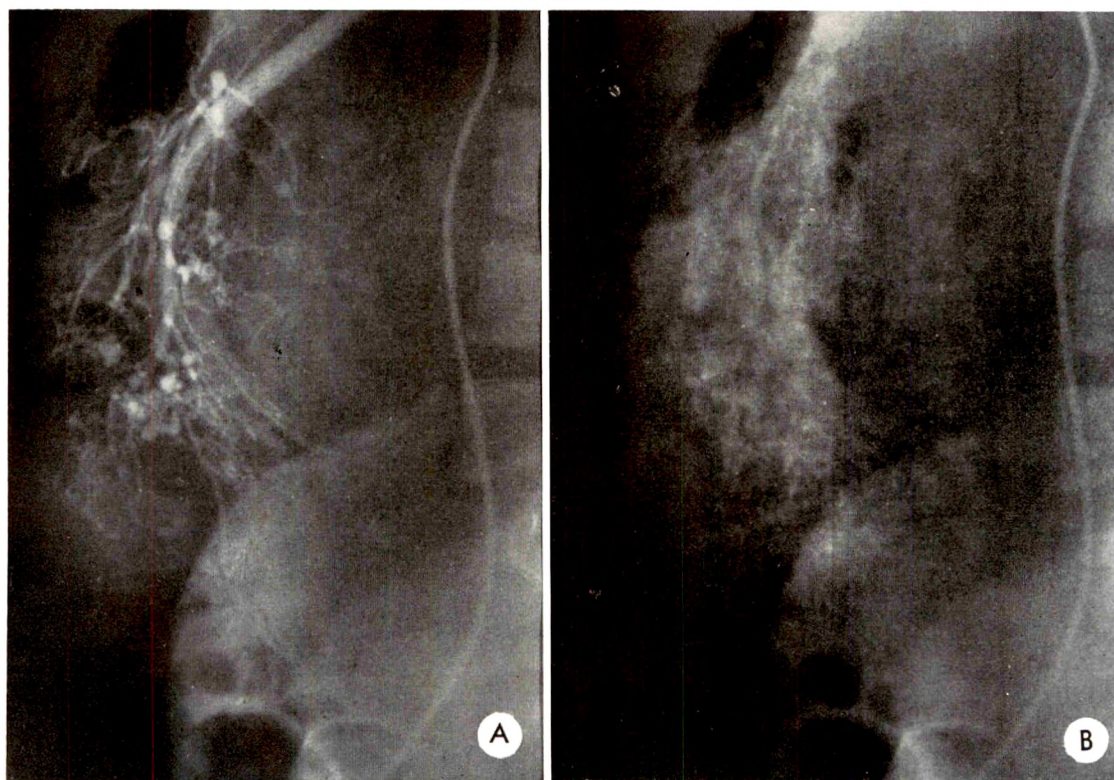


FIG. 6. (*A* and *B*) Lateral studies of the selective right renal arteriography showing similar findings to those in Figure 5, *A* and *B*. Note asymmetric involvement of the right kidney with avascular portion in the posterior half.

arteriograms (Fig. 7) showed stretched right hepatic arteries supplying an enlarged right lobe of the liver. None of the other organs examined showed evidence of angiomas. Roentgenograms of the skull revealed periventricular calcification, a plaque of calcium in the occipital

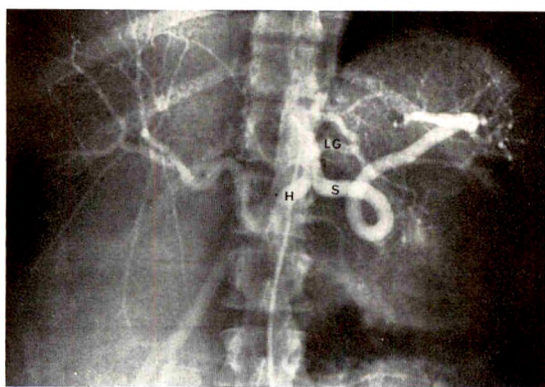


FIG. 7. Selective celiac arteriogram demonstrating spreading of the right hepatic arteries due to the presence of hepatic hamartomas. H=hepatic artery; S=splenic artery; LG=left gastric artery.

region, and calcification of the left basal ganglia (Fig. 8, *A* and *B*). Disseminated, rounded, small, osteosclerotic densities were uniformly distributed in the bony pelvis (Fig. 4, *A* and *B*). Cortical bone defects were observed in several phalanges of the hands and feet (Fig. 9, *A* and *C*). Localized areas of cortical thickening were present in some of the long tubular bones, especially the tibia and fibula (Fig. 9*B*). The chest roentgenogram showed elevation of the right hemidiaphragm and very fine honeycombing of the lower lobes (Fig. 10).

The patient was initially treated with chloramphenicol to which her urinary tract infection responded promptly. She was then explored with the hope of finding cystic lesions that could be decompressed to improve her kidney function.

At surgery, the liver and spleen were found to be enlarged approximately twice normal size, with the liver extending down to the level of the umbilicus. The right kidney was approximately 35 cm. long and 22 cm. wide. It appeared solid, yellowish-red and lobulated with



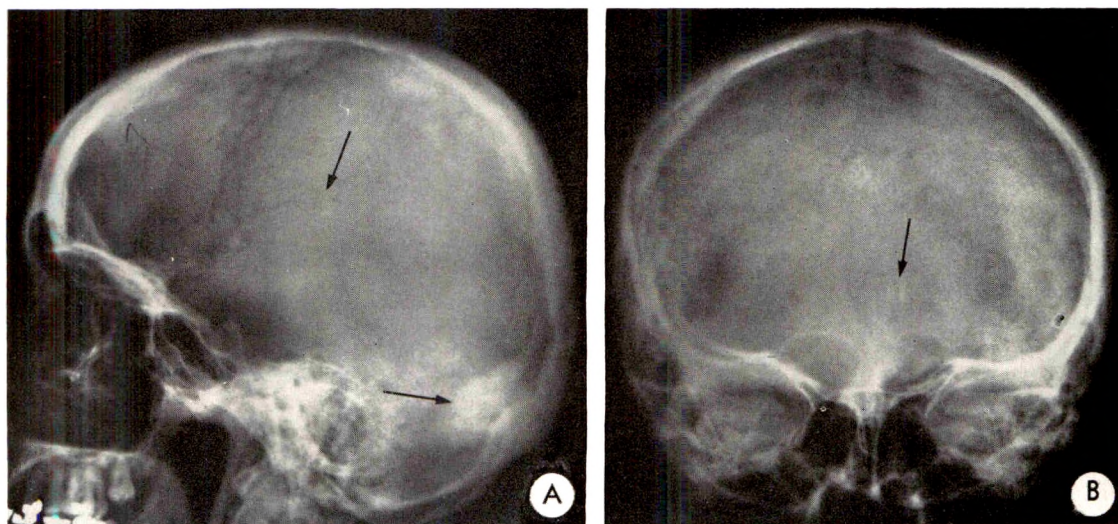


FIG. 8. Lateral (A) and frontal (B) roentgenograms of the skull demonstrating calcified plaques (lower arrow in A), periventricular calcification (upper arrow in A) and calcification in the region of the left basal ganglia (arrow in B).

no evidence of actual cysts. Needles were passed through some of the tumor lobules but other than blood, no fluid was obtained. The left kidney was approximately 25 cm. long and 15 cm. wide. It presented as a large solid mass with a smoother surface and a more yellowish appearance than the opposite kidney. The surface seemed thinly encapsulated. Bilateral renal biopsies and a liver biopsy were taken. The postoperative course was completely unremarkable and the patient was discharged 9 days after surgery.

The histopathology of both kidney biopsies was similar. The specimens were composed of mature adipose tissue, smooth muscle, and unusually thick-walled blood vessels of arteriole or small artery size (Fig. 11). The smooth muscle seemed to surround these blood vessels and often to fill in the intervening areas. The blood vessel walls were often hyalinized or partially replaced by connective tissue. The liver showed

several portal tracts which were markedly enlarged. There was marked fibrosis of these portal tracts without any extension into the surrounding tissue. The hepatic veins appeared considerably dilated. One of these large portal areas also showed a small circumscribed area of mature adipose tissue and a focus of proliferat-

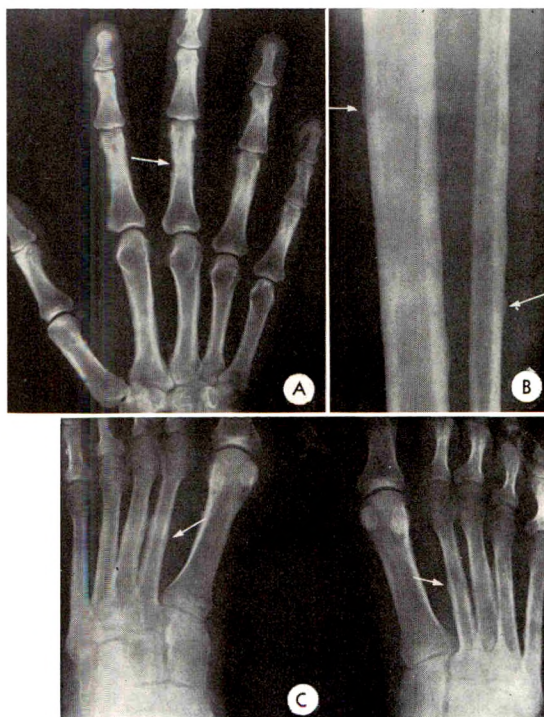


FIG. 9. Roentgenograms of the right hand (A), right leg (B) and feet (C). Note punched out cortical defects in the proximal phalanx of the second and third fingers, localized cortical hyperostosis in the tibia, fibula, metacarpals and metatarsals (arrows). The latter findings are identical to those seen in Gardner's syndrome.



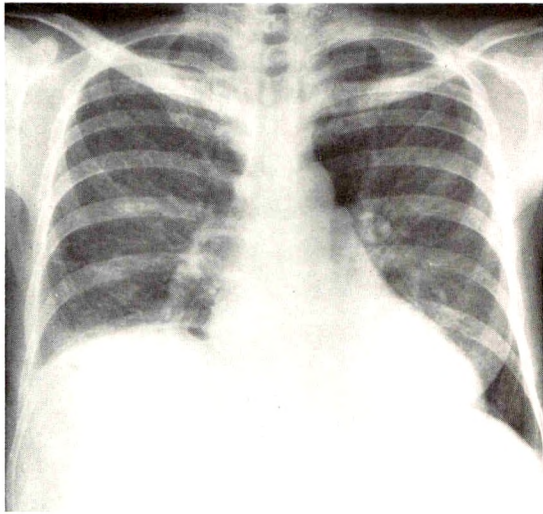


FIG. 10. Follow-up chest roentgenogram demonstrating elevation of the right hemidiaphragm and very fine reticular pattern in both lower lobes.

ing blood vessels of capillary size (Fig. 12). Histologic diagnosis of both kidneys was angiomyolipoma; and of the liver, focal portal hamartomas of mixed type.

#### DISCUSSION

Von Recklinghausen<sup>30</sup> in 1862 was the first to describe patchy sclerosis of the cerebral cortex, with his case also having multiple myomata of the heart. Bourneville<sup>6</sup> first applied the name "tuberous sclerosis" to these cerebral changes when he described 10 cases in a series of papers published during the years 1880-1900. This syndrome frequently bears the eponym "Bourneville's disease." The term "epiloia" was introduced by Sherlock<sup>32</sup> in 1911 to indicate the commonly encountered triad of adenoma sebaceum, epilepsy and mental deficiency. Moolton<sup>26</sup> in 1942 suggested the name "tuberous sclerosis complex" since the term tuberous sclerosis applied only to one feature in this disorder of multiple abnormalities. In the fully developed syndrome, numerous and extraordinary diversity of lesions may be encountered in the brain, kidneys, bones, skin, heart and lungs. Many of these anomalies belong to the category of hamartoma. This term was proposed by Albrecht<sup>2</sup> to characterize benign



FIG. 11. Photomicrograph of renal angiomyolipoma. Note mature adipose tissue, thick-walled small blood vessels and smooth muscle cells surrounding and between the vessels. These abnormal small arteries are a representative sample of those visualized by angiography. (Masson's trichrome  $\times 78$ .)



FIG. 12. Photomicrograph of hepatic portal hamartoma. Note marked portal tract enlargement by fibrous tissue, area of adipose tissue (left arrow) and focal aggregation of tiny blood vessels (right arrow). (H & E stain  $\times 44$ .)

tumor-like malformations composed of tissue indigenous to the parent organ, but abnormal in arrangement, quantity, or degree of maturation. The histopathologic alterations of the tuberous sclerosis complex have been well reviewed elsewhere,<sup>26,39</sup> so only a brief résumé of the syndrome findings will be presented.

*Cerebral lesions* may be of gross or microscopic size, and affect mostly the periventricular region of the cerebral cortex and the basal ganglia. Various combinations of hyperplastic astrocytic elements, dense glial tissue and hypervascularity are found. Calcification may develop around the blood vessels. Neurologic manifestations are very frequent and include epilepsy, mental retardation or both. In a much smaller number of patients, no clinical symptoms are present; sometimes even when cerebral lesions are found at autopsy. Frank neoplasia develops in a small percentage of cases.<sup>26</sup>

*Retinal lesions* are known as phacoma and consist of elevated nodules or plaques composed of glial tissues similar to those of the central nervous system.

*Skin anomalies* take several forms. The most common is the so-called "adenoma sebaceum" found in about 50 per cent of patients. These are groups of nodules occurring most often near the nose which histologically show either increase in dermal collagen and hair follicles or less commonly, a localized proliferation of the sebaceous glands.<sup>22</sup> These are rarely present at birth and may not appear until puberty. Less often found are thickened skin areas known as "shagreen patches," usually near the sacrum, and localized skin hyperpigmentation known as "café-au-lait" spots. Small fibrous tissue nodules called ungual fibromas are sometimes present, located either below or beside the fingernails or toenails. Some consider these equally important to adenoma sebaceum for the clinical diagnosis.

*Cardiac lesions* are represented by peculiar tumors called rhabdomyomas which are localized areas of abnormal myocardial

fibers distended with glycogen. These foci may be single or multiple, microscopic or large; they are associated with a very high mortality during infancy and childhood, even when clinically silent. Rhabdomyomas when single may occur in the absence of demonstrable tuberous sclerosis, although they are usually associated with the disease when multiple.<sup>39</sup>

*Hepatic hamartomas* have been described in 2 previous cases of tuberous sclerosis.<sup>15,27</sup> They may be lipomas, portal fibrolipomas or portal bile duct proliferation.

*Pulmonary lesions* have been reported in a few cases<sup>8</sup> and consist of many small cysts or areas of supporting tissue overgrowth.

*Bone changes* include patchy localized densities in the skull, vertebral column and pelvis which may simulate osteoblastic metastasis or osteopoikilosis.<sup>4,26</sup> In addition there may be small, cyst-like radiolucent areas in the phalanges.<sup>14</sup> Histologically, these are defects filled with dense fibrous tissue and surrounded by irregular bone proliferation.<sup>9</sup> Localized periosteal thickening may occur along the shafts of long bones, most commonly the phalanges.<sup>14</sup> They can simulate the cortical exostosis of Gardner's syndrome. We have found only 1 case report of tuberous sclerosis that showed the peculiar condition of "rheostosis," characterized by an irregular hyperostosis running down the shaft of a bone and confined to one limb.<sup>26</sup>

*Renal lesions* are most common next to cerebral ones, being found in 50 to 80 per cent of tuberous sclerosis patients.<sup>19,26,39</sup> These are usually characterized as mixed mesodermal tumors, being composed of adipose tissue, smooth muscle and blood vessels in varying proportions. In the overwhelming majority of cases, these lesions have proved benign; it is this fact, as well as their association with others of the varied nonepithelial, tumor-like malformations of the tuberous sclerosis complex, which has led to their classification as hamartomas.<sup>27</sup> There have been 3 cases reported with extension into the renal vein without further spread,<sup>2,13,27</sup> and 8 cases of "sarcoma" with



metastasis,<sup>3,7,26,36,39</sup> including 5 from the German literature,<sup>36</sup> in which the morphologic appearance suggested development from a pre-existing benign tumor. However, it is difficult to evaluate some of these reports, since "metastasis" in certain situations might represent hamartomas in other organs.

Histologically, the components of these tumors lead to the designation angiomyolipoma. The fatty elements are usually mature adipose tissue. The smooth muscle component may be sparsely or rather highly cellular. The nuclei are usually unremarkable but sometimes may display a considerable degree of anaplasia which uncommonly even suggests sarcoma. This cytologic variation accounts for much confusion in the literature regarding malignancy. In some cases the smooth muscle seems to originate from the walls of blood vessels, streaming out in a "pinwheel" fashion. The blood vessels are usually of small artery size, although varying in diameter. They are tortuous and often arranged in groups. Their wall is unusually thick for the diameter of the lumen, and it is common for most of the vessel wall to be hyalinized or partially replaced by connective tissue. Normal elastic tissue membranes are usually absent. They have been considered vascular malformations and seem more arterial in nature than venous. It is this phase of the renal lesion which angiograms in the present case demonstrated so beautifully.

In each kidney they may be single or multiple. When single, they are most frequently unilateral; when multiple, they are generally bilateral. Single and unilateral kidney hamartomas usually occur in the absence of demonstrable tuberous sclerosis. One hundred and three cases of renal angiomyolipoma without tuberous sclerosis have been reported.<sup>1,3,11,13,18,19,20,25,29,31,32,34,38</sup> In the last 5 years we have seen 2 at our institution which were surgically removed, with no stigmata of tuberous sclerosis reported.<sup>16,17</sup> Unfortunately, it is difficult to

evaluate many of the reported cases, since a patient may have histologic stigmata of tuberous sclerosis not evident clinically, or which even at postmortem examination might conceivably be overlooked. Some authors feel that renal hamartomas which occur in patients without the usual stigmata of tuberous sclerosis may represent the so-called "forme fruste" of that syndrome.<sup>2,13,29</sup>

The great majority of these tumors are discovered only at autopsy. They may be of microscopic size or grossly visible. They are most commonly small, most often less than 2 cm. in diameter, but have been reported as large as 20 cm. in diameter. Those which are clinically evident display symptoms of infection, pain, or hematuria—the latter due to the abnormal tumor blood vessels which may cause hemorrhage. Of 30 patients described by Price and Mostofi,<sup>29</sup> 3 presented as acute abdominal emergencies. At the other end of the clinical spectrum, a tumor may rarely come to attention as an asymptomatic abdominal mass.

Urograms in these patients usually show a space-occupying lesion distorting and displacing the pelvis and calyces according to its location and size. These masses are frequently difficult to distinguish from large cysts or renal carcinoma. Lucent areas corresponding to the fatty tissue component may be helpful if present.<sup>18</sup> Angiomyolipomas of the kidney and polycystic disease may have a very similar pyelographic appearance in some cases.<sup>35</sup> The association of tuberous sclerosis and polycystic renal disease has been reported in 5 cases.<sup>15,19,27,28,35</sup> Arteriography has been employed in 6 reported cases of angiomyolipoma during preoperative work-up.<sup>10,19,20,27,38</sup> In 5 a translumbar technique was used, and in 1, the Seldinger catheter method.<sup>20</sup> The findings and their interpretation are summarized by Khilnani and Wolf as follows,<sup>18</sup> "Puddling of the opaque material as well as vascular displacements were demonstrated which resembled the findings seen with hypernephroma." These authors were led to

conclude: "It would seem, therefore, that this is not a useful technique for differential diagnosis in such instances."

We feel that in our case of angiomyolipoma, there were changes which are characteristic and which are not found in either hypernephroma or polycystic disease. In fact, the diagnosis of tuberous sclerosis was first considered only when the selective renal studies were evaluated, with the other stigmata mentioned earlier being uncovered in retrospect. Here, as in other lesions, the technique of selective renal angiography usually offers better delineation of the kidney architecture than translumbar aortography. The normal renal angiogram demonstrates three phases: arterial, capillary (nephrographic), and venous. The initial arterial phase occupies about 2 seconds. The renal arteries, the arcuate and interlobar arteries, and, in many instances, the small interlobular arteries are demonstrated. There is a uniform branching pattern throughout. The arteries maintain a regular diameter and a uniform tapering appearance. The nephrographic phase lasts about 5 to 10 times the preceding arterial phase, although varying from patient to patient. The dense complex nephrographic opacification time period is usually followed by demonstration of faintly outlined renal veins, the venous phase. This is not always seen, and is superimposed on the nephrographic phase. The venous phase usually appears about 8 to 10 seconds after initial injection of contrast material and is of variable duration.

In renal carcinoma (hypernephroma), there are several distinguishing features. In the arterial phase, abnormal vessels are usually seen. Since renal carcinoma vasculature is no different from that of tumors elsewhere, there are both quantitative and qualitative abnormalities. There is hypervascularity within the tumor. The tumor vessels have abnormal distribution and branching patterns, are pleomorphic in respect to both size and outline, take aberrant courses, display focally dilated areas

which have an irregular contour and size, and lack normal tapering. In addition, the angiograms characteristically show pooling or "puddling" of the contrast medium within the tumor; this may appear anywhere in the tumor but most often at the periphery. They are of variable number and size, and have rounded or irregular shapes. These localized collections of contrast material are due to neoplastic vessel anastomoses resembling arteriovenous malformations, to hypervascular areas contrasted to contiguous avascular regions, or to the formation of "venous lakes" due to venous obstruction. They appear at the end of the arterial phase and persist, or only during the nephrographic stage, depending on the etiology. Finally, the venous phase in hypernephroma is often unusually rapid in onset, showing either normal or abnormal vessels.

In our case of angiomyolipoma, the arterial phase showed hypervascularity with abnormal vessel distribution. In addition, some of the morphologic aspects of hypernephroma vessels were present, such as loss of normal tapering and presence of unusual tortuosity. However, what was most striking was the presence of many peculiar, small, regular outpouchings resembling berry aneurysms from the interlobar and interlobular arteries (Fig. 5, *A* and *B*). In some places they seemed to terminate the interlobular arteries, with an appearance suggesting a cluster of grapes. They were in definite contrast to the irregular size and contour manifested by the focal ectasia of hypernephroma vessels. These peculiar aneurysm-like structures were best seen in the arterial phase. During the nephrographic phase, they were obscured by the superimposed nephrographic effect, and thus became an irregular pooling with asymmetric distribution which would be indistinguishable from the "puddling" of hypernephroma. The venous phase developed normally, again in contrast to the early appearance often associated with carcinoma.



Polycystic renal disease could be easily ruled out, since it has a relatively avascular angiographic pattern.

## SUMMARY

A classic case of the tuberous sclerosis complex is presented. Selective renal angiographic studies disclosed clear differentiation between large renal hamartomas and the possibility of polycystic kidney or renal carcinoma. In addition, angiography was suggestive of hepatic involvement.

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## PULMONARY MANIFESTATIONS OF TUBEROUS SCLEROSIS\*

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**T**UBEROUS sclerosis, although primarily considered a cause of mental retardation, affects many organs other than the central nervous system. The manifestations in the brain, kidney and bones are not uncommon; however, the pulmonary changes are much less frequent. Few patients with pulmonary changes have had postmortem studies. Accordingly, we are presenting a case report of a patient with extensive, and rather unusual, pulmonary lesions with postmortem findings.

### REPORT OF A CASE

This 24 year old white woman was referred to the University of Arkansas Medical Center with a history of a 20 to 30 pound weight loss over the past 6 months. Hematuria had been present for 3 weeks prior to admission. An older sister stated that the patient had seizures during the first 2 years of life but none subsequently. Because of mental retardation, she was unable to attend school but cared for herself reasonably well. Nine siblings were described as normal by the sister with specifically no history of mental retardation.

The patient appeared chronically ill. Mental deficiency was obvious but she could answer simple questions. A fine nodular rash overlay the skin of the nose and around the chin. Small, brown, cutaneous masses were present over the right eyelid and the outer margin of the left canthus. Examination of the retinae was inadequate. Rales and rhonchi were present over both lung fields. The liver was moderately enlarged. An ill-defined mass without a definite lower border was present in the left upper quadrant.

The extremities were normal; specifically, no ungual fibromas were present.

Other than mental retardation, the neurologic examination was within normal limits.

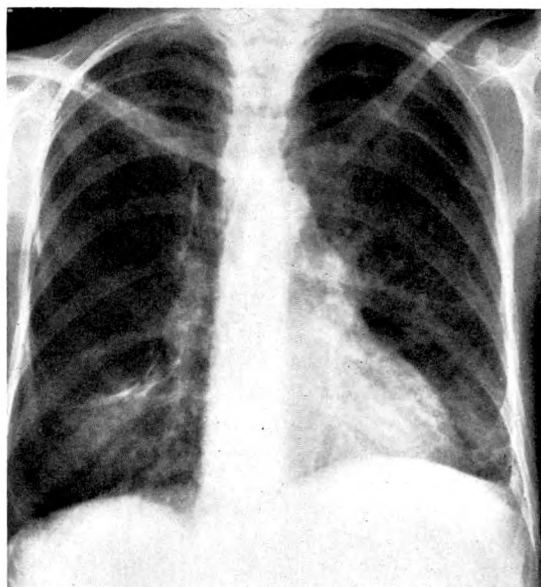


FIG. 1. Posteroanterior roentgenogram of the chest on the day of admission. The lungs are overexpanded with an increase in the anteroposterior thoracic dimension. Several large cystic areas, measuring as much as 7×7 cm., are present in the right upper lung field. A diffuse reticular pattern is present in both lower lung fields with an appearance of multiple small cysts, the so-called "honey-comb lung." The bones of the thoracic cage appear normal.

Hemoglobin was 9.0 gm. per cent. Two plus proteinuria and 8 to 12 leukocytes per high power field were present on examination of the urine. The blood urea nitrogen was 25 mg. per cent.

A diagnosis of tuberous sclerosis with moderately severe mental retardation was made.

The patient's condition remained good until the third hospital day when she suddenly developed severe left flank pain and hypotension. She expired within 1 hour in shock.

*Roentgenographic Findings.* The roentgenographic findings are shown in Figures 1 through

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FIG. 2. Excretory urogram performed on the second day after admission. Large masses fill the abdominal cavity. The kidneys function well but the calyceal systems are markedly stretched and elongated. The left calyceal system is displaced inferiorly, extending into the lower abdomen and pelvis.



5. We consider these findings characteristic of the osseous, renal and pulmonary manifestations of tuberous sclerosis.

*Postmortem Findings.* The peritoneal cavity contained approximately 350 cc. of blood, largely in the pelvis.

The left kidney filled the flank, measured  $30 \times 17 \times 15$  cm. and weighed 4.3 kilograms. The upper two-thirds of this kidney was replaced by a large, yellowish-tan, irregularly lobulated tumor extending through the cortex. Within the tumor were extensive areas of old and recent hemorrhage. Similar but smaller tumor masses were present in the right kidney. The perirenal tissues were not invaded by the neoplasm.

On microscopic examination, the renal tumors

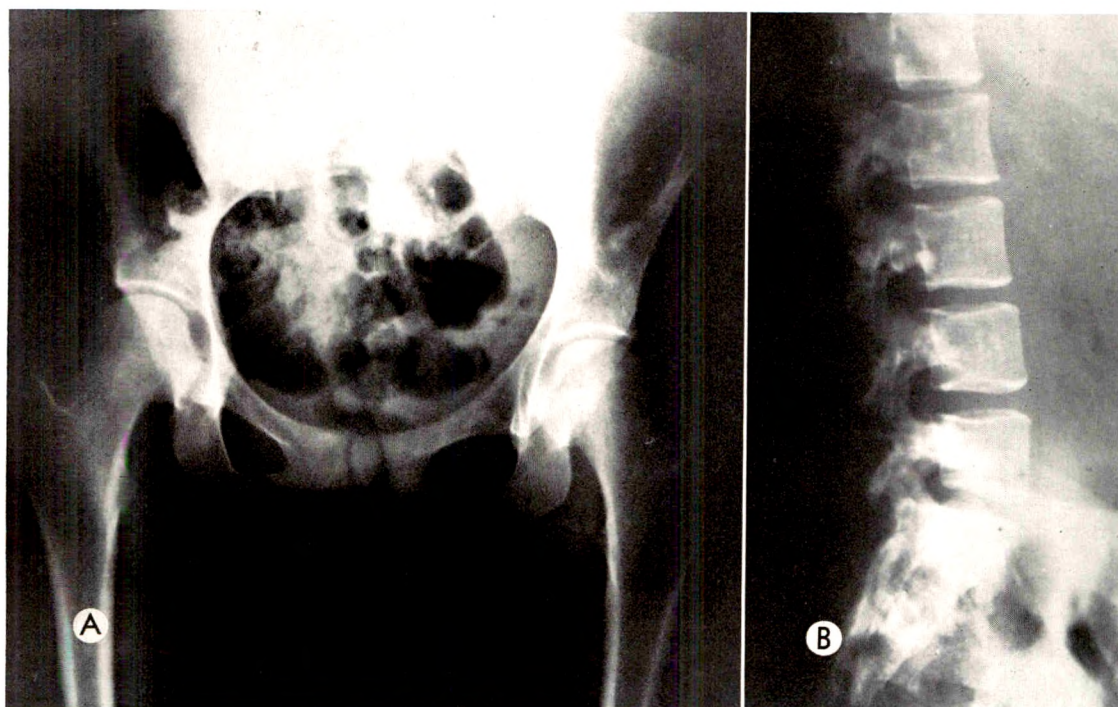
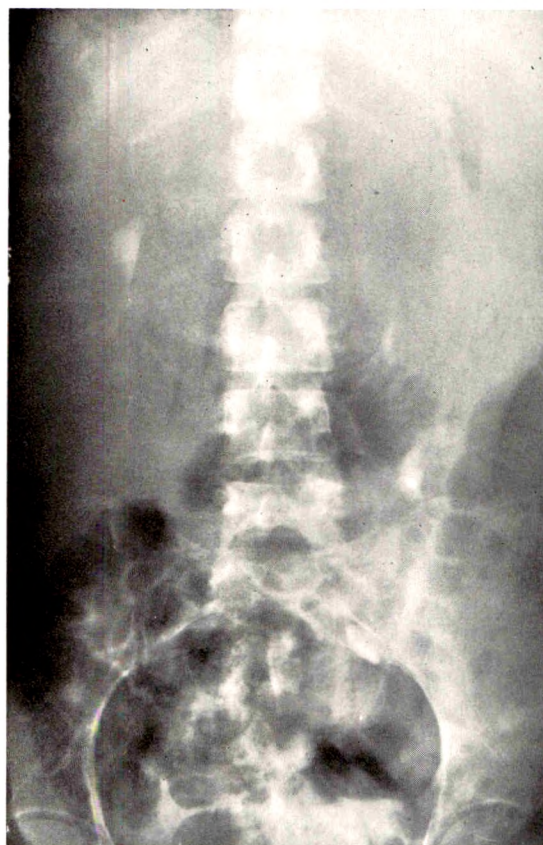


FIG. 3. (A and B) Pelvis and lumbar spine. Small ill-defined areas of increased density are present throughout the bony pelvis and in the vertebral bodies of the lumbar spine.





FIG. 4. Feet. There is cortical thickening with wavy margins in the shaft of the right second metatarsal. A well-healed fracture is present in the shaft of the right fifth metatarsal.

were found to contain large numbers of pleomorphic spindle cells with admixed islands of mature fat cells. Angiomatous areas containing thick-walled, blood-filled vessels were also conspicuous. The tumors were sharply demarcated from the normal parenchyma. No blood vessel invasion was apparent.

Scattered throughout both lungs were innumerable small cysts measuring 1 to 8 mm. in diameter (Fig. 6). The cysts contained air, but, grossly, did not appear related to the branches of the tracheobronchial tree; however, on microscopic examination, some of the cysts were found to communicate with small bronchioles. Large subpleural blebs were present over the lateral aspect of the right upper and right middle lobes. The pleural space was obliterated by dense, fibrous adhesions.

Microscopic examination revealed that most of the small cysts had no apparent epithelial lining although some were lined in part by cuboidal and columnar epithelium. The walls of some cysts contained bands of smooth muscle

cells. The cysts were bounded by distorted alveolar septa and atelectatic alveoli. Many alveolar spaces were lined by metaplastic cuboidal cells. Many alveoli were filled with hemosiderin-laden macrophages and a few contained edema fluid. Some alveolar septa were thickened by heterogenous cells having round, oval or elongated nuclei. The cells with round nuclei resembled pulmonary macrophages, similar to the macrophages seen in the alveolar spaces.

The gyri of the cerebral hemispheres contained multiple firm nodules measuring 1 to 1.5 cm. in diameter. A 1 cm. in diameter nodule was found in the right caudate nucleus and similar smaller nodules were present beneath the ependyma of both lateral ventricles. Microscopically, the cortical lesions displayed gliosis, increased number of astrocytes and disorientation of pyramidal cells. The subependymal lesions were characterized by nests of large polyhedral cells with eccentric nuclei surrounded by swarms of glial fibers. The largest of these contained microscopic foci of calcification.

Small hamartomata were found in the thyroid and liver as well as in the gastric mucosa. No cardiac tumors were present. Attempts to biopsy the sclerotic areas in the lumbar vertebrae were unsuccessful.

The pathologic diagnosis was tuberous sclerosis with widespread hamartomata. The cause of death was acute, massive intra-abdominal hemorrhage from the tumor mass in the left kidney.



FIG. 5. The skull is normal. Specifically, there are no intracranial calcifications or sclerotic areas in the bony tables.

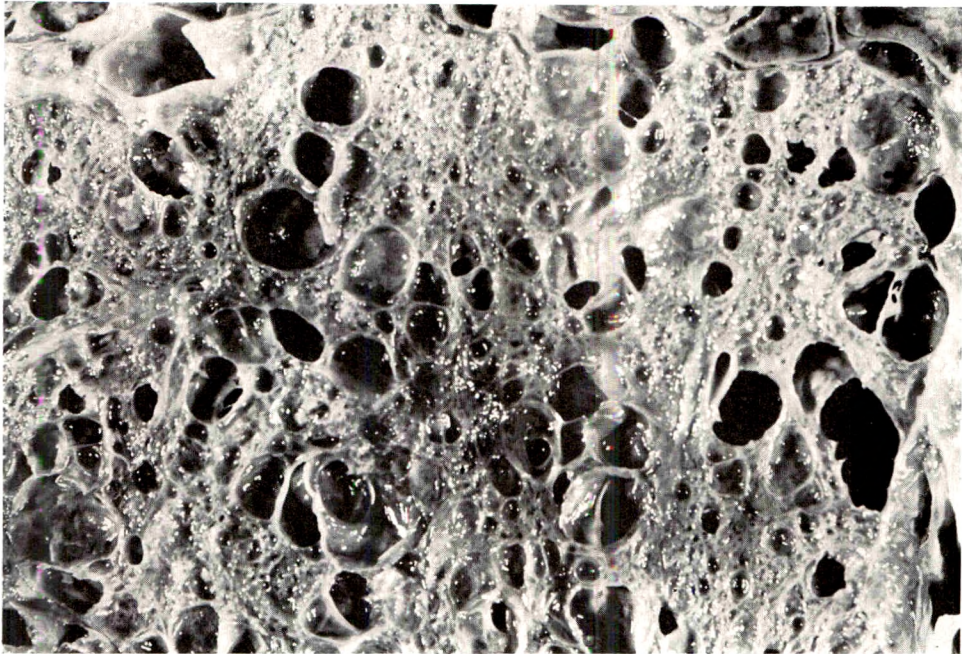


FIG. 6. Photograph of the cut gross lung specimen at autopsy. Multiple cysts of varying size are scattered throughout the entire lung.

#### DISCUSSION

The roentgenographic manifestations usually described are those which mirror the common pathologic changes; intracranial calcifications, sclerotic areas in the bony tables of the skull and intrarenal masses. However, other organ systems may also have roentgenographically demonstrable changes.

The roentgenographic manifestations fall into 4 general areas:

1. Cerebral: intracranial calcifications and pneumographic evidence of intraventricular tumors, with ventricular obstruction in some patients, are often present.<sup>11</sup>
2. Bony: patchy areas of increased density in the skull, vertebrae, pelvis and long bones may be present. These often mimic osteoblastic metastases.<sup>11</sup> Subperiosteal new bone formation and cyst-like lesions are found in the metatarsals, metacarpals and phalanges.<sup>4, 8, 10, 14</sup>
3. Visceral: hamartomas are usually found in the kidneys<sup>7</sup> but have been reported in almost all organs.
4. Pulmonary: pulmonary involvement was first described by Berg and Vejens<sup>2</sup>

in 1939 with a second article by Berg and Zachrisson<sup>3</sup> appearing in 1941. Dawson<sup>6</sup> has extensively reviewed the clinical aspects of pulmonary tuberous sclerosis. Presenting symptoms in his cases were spontaneous pneumothorax, pulmonary insufficiency and cor pulmonale. Although the skin and cerebral features are noted usually in childhood, the pulmonary manifestations are recognized later. The youngest patient described by Dawson with symptoms referable to the lungs was 30 years old. Dyspnea had been present from 1 to 11 years in his 8 fatal cases. Ackermann,<sup>1</sup> in 1944, described a patient with an interstitial pulmonary infiltrate and bone lesions. In the light of present knowledge, this case almost certainly represents histiocytosis X rather than tuberous sclerosis.

Roentgenographically, the lung changes are those of interstitial infiltration. The pulmonary changes are rarely associated with intracranial calcifications but bone changes are almost universally present.<sup>6</sup> Furthermore, the lung changes may be present in a patient with incomplete clinical manifestations of the disease. The differen-



tial diagnosis roentgenographically lies among those diseases causing a diffuse interstitial infiltrate.<sup>12</sup> Histiocytosis X, sarcoid, scleroderma and idiopathic interstitial fibrosis are the more common processes presenting a similar roentgenographic appearance.

Early changes are a reticular infiltrate which progresses to "honeycombing" (the term is used here in a radiologic rather than pathologic sense). Spontaneous pneumothorax is common. Large cysts, as present in this patient, are uncommon. Bronchographies have been done, but filling of the cystic spaces has not been demonstrated in tuberous sclerosis.<sup>6</sup> This is in marked contrast to polycystic lung or bronchiolar dilatation with muscular hyperplasia.<sup>5</sup>

The pathogenesis of the lung changes is obscure. Most of the patients described with lung changes have shown findings similar to our patient in that multiple cysts are present, separated by areas of fibrosis and particularly characterized by hyperplasia of the smooth muscle cells in the alveolar walls.

The relationship of tuberous sclerosis to so-called muscular hyperplasia of the lung existing as an independent entity is not clear.<sup>9,13</sup> It would appear, however, that these cases do not represent an incomplete manifestation, or forme fruste, of tuberous sclerosis.

#### SUMMARY

A patient with the typical clinical, roentgenographic and pathologic features of tuberous sclerosis is reported. Pulmonary involvement was extensive, a rare finding in tuberous sclerosis. The pulmonary involvement is indistinguishable from other causes of interstitial fibrosis. However, bony changes of sclerosis, periosteal new bone formation and cysts are almost invariably associated with the pulmonary lesions. Conversely, intracranial calcifications are rarely associated with pulmonary

involvement. The complete clinical syndrome may not be present in all patients with pulmonary involvement.

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## ROENTGENOGRAPHIC FINDINGS IN THE RENAL LESION OF TUBEROUS SCLEROSIS

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THE entity known as tuberous sclerosis was first described by Bourneville in 1880 as a triad of epilepsy, mental retardation and adenoma sebaceum. The name derived from curious tuber-like neuroglial proliferations in the cerebral cortex and subependymal areas of the ventricles. Since that time, the disease has been further defined in all its ramifications in the literature as an inherited disorder exhibiting a varied spectrum of severity and manifesting multiple hamartomatous lesions in various organ systems.

In recent months, 2 striking instances of renal involvement in tuberous sclerosis have been encountered which evidenced sufficiently characteristic roentgenographic signs that the diagnosis could be suggested with considerable confidence. These features of renal involvement in tuberous sclerosis have been little emphasized in the radiologic literature in the past and are felt to be worthy of report.

### REPORT OF CASES

CASE I. R. R., a 36 year old white female, was first admitted to Overlook Hospital with a chief complaint of abdominal pain.

One month prior to admission, she had been kicked in the abdomen by her mentally retarded 6 year old son while dressing him. There appeared subsequent increasingly severe right-sided abdominal pain, urinary frequency and constipation. Two weeks prior to admission, a right lower quadrant mass was palpated which increased slightly in size. In the 2 days prior to admission, flecks of blood were noted in the urine.

At 3 years of age, the patient had had her first convulsive seizure; this did not recur until her sixth year, after which time the seizures occurred sporadically until the age of 12 years, when they ceased and never recurred, without the use of medication. The diagnosis of tuberous sclerosis was first made at the Neurological

Institute of New York at the age of 6 years. She is of average intelligence and had completed high school without difficulty. Her parents were free of the disease, but a maternal grandmother had had an angiomyolipoma of the kidney removed in 1948 at 81 years of age. One mentally retarded cousin of her mother's is known to be alive in his sixth decade. The patient's 6 year old son had been under care since shortly after birth when generalized seizures began. He is severely retarded and now is institutionalized with an established diagnosis of tuberous sclerosis.

On physical examination, an 8×10 cm. smooth, tender, firm, movable mass was felt. The left kidney felt enlarged as well. Multiple sebaceous adenomas of the face were noted.

Urinalysis showed a 1+ albuminuria and innumerable red blood cells per high power field. The blood urea nitrogen was 14.5 mg. per cent, and phenolsulfonphthalein excretion was 7 per cent in the first hour and 17.5 per cent in the second hour.

Excretory urograms (Fig. 1) and nephrotomograms (Fig. 2) demonstrated enlarged mottled kidneys with pelvocalyceal distortion and multiple lucencies in the kidney parenchyma. Skull roentgenograms (Fig. 3) demonstrated intracerebral calcification and chest roentgenograms (Fig. 4) showed the pattern believed characteristic of tuberous sclerosis.

At laparotomy, a very large mass was found to be present at the lower pole of the right kidney. On frozen section, this was felt to be a liposarcoma, and a right nephrectomy was done.

The right kidney weighed 584 gm.

The serosal surface was irregularly mottled and yellow-brown in color. On cut section, there were numerous greasy yellow tumors scattered throughout the parenchyma, replacing at least 75 per cent of it, the masses measuring from 0.1 to 6.5 cm. in dimension (Fig. 5). A large hematoma was present in the lower pole.

The microscopic sections were reviewed by a number of pathologists, their opinion in consensus being that this was a benign angiomyo-





FIG. 1. Case 1. Excretory urogram. The renal outlines are enlarged, ill-defined and mottled by innumerable lucencies. Pelvocalyceal distortion of a type similar to polycystic disease is seen. Note multiple areas of increased osseous density in the pelvis and vertebral pedicles.

lipoma, despite a distinct increase in lipoblastic activity, which had led to the frozen section diagnosis of liposarcoma.

The patient recovered uneventfully, and was discharged on the 11th postoperative day. She has had no further difficulty.

CASE II. V. F., a 55 year old white unmarried female librarian, was admitted to Overlook Hospital for the first time with a chief complaint of hematuria.

Two weeks prior to admission, she had noted the sudden onset of total painless hematuria, without clots. There was no dysuria, urgency, frequency or flank pain. Cystoscopic examination demonstrated blood coming from the right ureteral orifice. No bladder lesion was seen. Roentgenographic studies elsewhere (Fig. 6 and 7) had demonstrated a mass lesion in the upper pole of the right kidney, deforming the right collecting system.

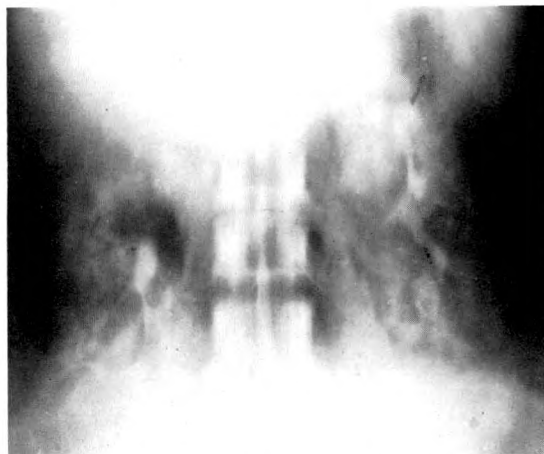


FIG. 2. Case 1. Nephrotomogram. Pericalyceal and parenchymal fat distribution is evident in both kidneys, the lesions varying greatly in size.

There was no history of nervous or mental disease; no family history of such illness could be elicited. Both parents had died in their eighth decades of unrelated illness.

On physical examination, no masses could be felt; there were no significant physical findings save for multiple facial sebaceous adenomas. Additional roentgenographic study was not undertaken in this instance.

On August 10, 1964, a right nephrectomy was done. A 6 cm. lobular tumor replaced the upper pole of the right kidney, its cut surface exhibiting a rubbery, yellowish-gray appearance, with focal areas of congestion. On microscopic examination, the lesion proved to be an angiomylipoma.

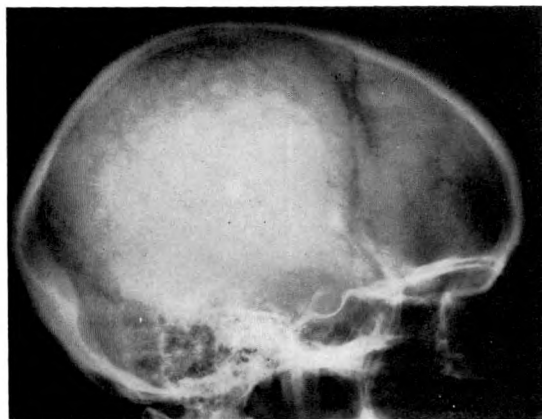


FIG. 3. Case 1. Multiple calcifications are noted in the brain, presumably in the wall of the lateral ventricles.



FIG. 4. Case 1. Detail of left lung. The classic reticular distribution of the lesions of tuberous sclerosis in the lung is well demonstrated.

The patient has remained well since, without recurrence of urinary tract symptoms.

#### DISCUSSION

The incidence of renal hamartomas or angiomyolipomas has been variously estimated at from 50 to 89 per cent of instances of tuberous sclerosis; conversely, it is estimated that 50 per cent of all renal hamartomas occur in patients with tuberous sclerosis.<sup>17,18,19</sup> Khilnani and Wolf<sup>17</sup> reported 6 cases of renal hamartolipoma in which there was no associated tuberous sclerosis, while the urologic literature is replete with reports of angiomyolipomas of



FIG. 5. Case 1. Cut section of the right kidney shows the multiple fat bearing lesions of the kidney, with a recent area of hemorrhage in the lower pole.

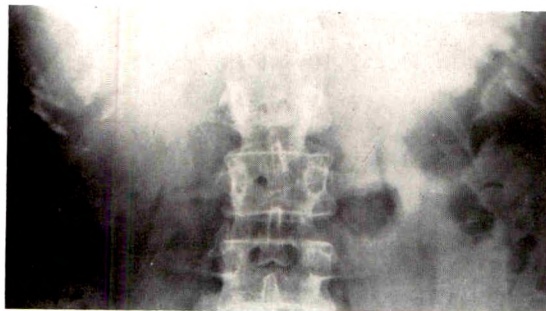


FIG. 6. Case II. A preliminary plain roentgenogram shows suspicious radiotranslucency in the upper pole of the right kidney and expansion of the upper pole.

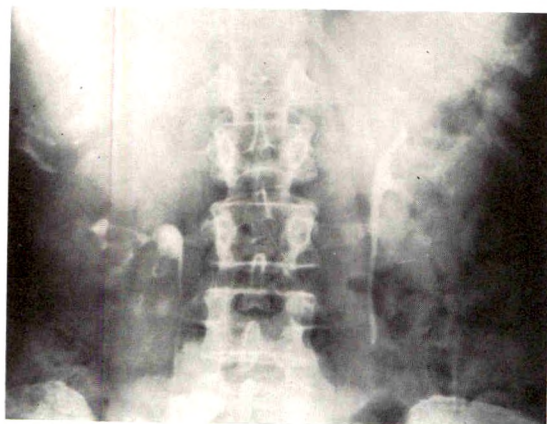


FIG. 7. Case II. Excretory urogram. Downward displacement of the right collecting system is noted. The lucent character of the tumor becomes more obvious and is less readily confused with intervening densities.



the kidney, the great majority of which occurred in instances of tuberous sclerosis.  
<sup>2, 8, 11, 12, 15, 20, 21, 25</sup>

The tumors are composed of blood vessels, smooth muscle and adipose tissue, and have been variously classified as angioliipomas, myoliipomas or angiomyoliipomas, depending upon the predominant histologic elements; they vary from microscopic to enormous size and are frequently bilateral in tuberous sclerosis.<sup>18</sup> Growth of the lesions is slow; occasional episodes of infarction and hemorrhage may result in their sudden enlargement and in hematuria or flank pain, prompting urgent clinical investigation. Malignant degeneration is said to be rare.<sup>6, 17, 27</sup> Reports of liposarcomas of the kidney<sup>9, 10, 26</sup> have been questioned in recent years<sup>17</sup> as to their accuracy in view of long follow-up on certain cases. One such case of liposarcoma of the kidney in a young woman afflicted with tuberous sclerosis was reported by Fish and McLaughlin<sup>9</sup> in 1946. This case has been personally reviewed by the author and verified as having later died of liposarcoma involving the retroperitoneal space 5 years after the initial report, the postmortem slides having lately been reviewed by several pathologists. Indeed, there was considerable concern about the tissue diagnosis in Case 1 where much lipoblastic activity was noted. The consensus of opinion among several consultants was that this was a mesenchymal tumor similar to those described by Stout<sup>24</sup> but of a benign nature, best fitting the classification of angiomyoliipoma.

#### ROENTGEN FEATURES

On plain roentgen examination, the most striking finding is the presence of enlarged renal images in which one or more ill-defined radiotranslucent areas of fat density are seen. These result in a mottled appearance of the renal shadow which is usually ill defined in all or a portion of its border. On excretory urograms, the lesion may present as a mass displacing or distorting the renal collecting system (Fig. 7). More characteristic is an attenuation and

spreading apart of the calyces resembling the appearance of polycystic disease (Fig. 1). This appearance is due to the presence of multiple angiomyoliipomas in the renal parenchyma which are fat bearing.<sup>19, 23</sup> Tomographic and nephrotomographic study makes more clearly manifest the striking pattern of fat distribution in the kidney (Fig. 2). This has been shown in a prior instance by Emmett<sup>8</sup> in a solitary angiomyoliipoma, but the significance of the relative radiotranslucency of the lesion was misinterpreted as being due to infarction.

Other roentgenographic findings in tuberous sclerosis have been previously described in detail. Multiple areas of increased density in the skeleton, occurring in any bone including the vertebral bodies, pedicles and the skull, are believed to represent osteomas, again of a hamartomatous nature.<sup>1, 4, 7, 13, 14</sup> Much interest has been focussed on the pulmonary lesion in tuberous sclerosis;<sup>1, 3, 5, 22</sup> this is well demonstrated in Figure 3. The reticular appearance of the lung is due to smooth muscle and vascular proliferation delimiting multiple cystic areas lined by cuboidal epithelium. The focal neuroglial proliferations described as the earliest lesion may calcify and betray their presence (Fig. 4). These may become the sites of true gliomatous proliferation; if strategically situated, as in the periaqueductal area, they may cause death.<sup>16, 19</sup>

All or some of these lesions may be demonstrable roentgenographically. A number of clinical signs of interest is, of course, to be found; chief among these is the misnamed adenoma sebaceum of the facies, which is truly angiomyoliipomatous change in the skin. Shagreen patches, in various areas of the skin, retinal phacomias, subungual fibromas and occasional lenticular opacities are noted as well.

#### CONCLUSIONS AND SUMMARY

The presence of a tumor mass or masses of an ill-defined yet radiotranslucent nature in one or both kidneys, perhaps associated with pelvocalyceal distortion, or

the presence of multiple mottled lucencies in an expanded poorly defined renal image, the collecting system of which resembles that seen in polycystic disease, should suggest the presence of angiomyolipomatous change in the kidney parenchyma. Coupled with other roentgenographic signs in the lung, brain and skeletal system, these findings should indicate the presence of tuberous sclerosis.

Since many lesions present initially as a urologic problem, it may be possible to avoid a useless nephrectomy since malignant degeneration in these lesions is quite rare and the involvement is often bilateral. Nephrotomographic study is of considerable importance in clearly defining the structural alterations in the kidney.

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# INCREASED THICKNESS OF THE FIBULA IN DUCHENNE MUSCULAR DYSTROPHY\*

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**R**ECENT refinements of clinical, genetic and biochemical criteria have made classification of the muscular dystrophies more precise. The major subgroups are:<sup>10,11</sup>

1. Duchenne muscular dystrophy
  - (a) sex-linked recessive
  - (b) autosomal recessive
2. Limb girdle muscular dystrophy
  - (a) autosomal recessive
  - (b) sporadic
3. Facioscapulohumeral muscular dystrophy
  - (a) autosomal dominant

The Duchenne variety, also called progressive or pseudohypertrophic muscular dystrophy, is the most severe and frequent subgroup. It is usually inherited as a sex-linked recessive trait which is transmitted by asymptomatic female heterozygote "carriers" and which produces clinical involvement only in males. The disease begins early in childhood with clumsiness in walking, frequent falling and difficulty in climbing stairs. The disease progresses steadily, involving both lower and upper extremities, and leading to contractures of the Achilles tendon and the knee and elbow joints. Limb girdle dystrophy affects either sex, begins in the second decade or later, and usually follows a milder course. Facioscapulohumeral or Landouzy-Déjerine dystrophy involves the facial muscles at first and the shoulder and pelvic girdle musculature later.

## REVIEW OF THE LITERATURE

Since 1873, when Friedreich<sup>8</sup> described the first autopsy findings in an 18 year old boy with muscular dystrophy, skeletal deformities have been reported several times, including scoliosis of the spine, narrowing of

Diagnosis	No. of Patients
Muscular Dystrophy	
Duchenne	25
Limb Girdle	3
Facioscapulohumeral	1
Other Neurologic Disorders	19
Normal	51
Total	99

the shafts of long bones, rarefaction of the shafts and heads of long bones, hypoplastic scapulae, coxa valga, and short vertical rami of the mandible. The pathogenesis of these osseous abnormalities is unknown but until recently they were generally considered not to be due to muscle inactivity.<sup>1,2,4,5,7,8,9,14</sup> The finding of similar changes in the long bones of 4 cases of poliomyelitis and 2 of dermatomyositis have led Walton and Warrick<sup>12</sup> to suggest that the skeletal abnormalities are secondary to disuse, to absence of normal stress on the osseous attachments of muscles and to the postural deformities resulting from muscle weakness and contractures. In 1963 Kaufmann<sup>6</sup> reported an unusual widening of the fibula in its anteroposterior diameter in 9 patients with pseudohypertrophic muscular dystrophy, ages 7 to 17 years.

## MATERIAL AND METHODS

Ninety-nine patients are included in this survey (Table 1). The 29 patients with muscular dystrophy comprized 25 with the Duchenne type, 3 with limb girdle and 1 with facioscapulohumeral dystrophy. In all dystrophy patients anteroposterior and

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TABLE II  
MEAN VALUE OF FIBULA/TIBIA (F/T) RATIO

	Duchenne Dystrophy	Other Neuromuscular Disorders	Normal
F/T	0.77 ± 0.15	0.59 ± 0.11	0.58 ± 0.07
No. of Patients	25	19	51

lateral roentgenograms of both tibiae and fibulae, a posteroanterior roentgenogram of the chest and posteroanterior and lateral roentgenograms of the skull were made. Additional roentgenograms of other parts of the skeleton were taken when indicated clinically. Each patient had a history and physical examination, 12-lead electrocardiogram and serum creatine phosphokinase determination.

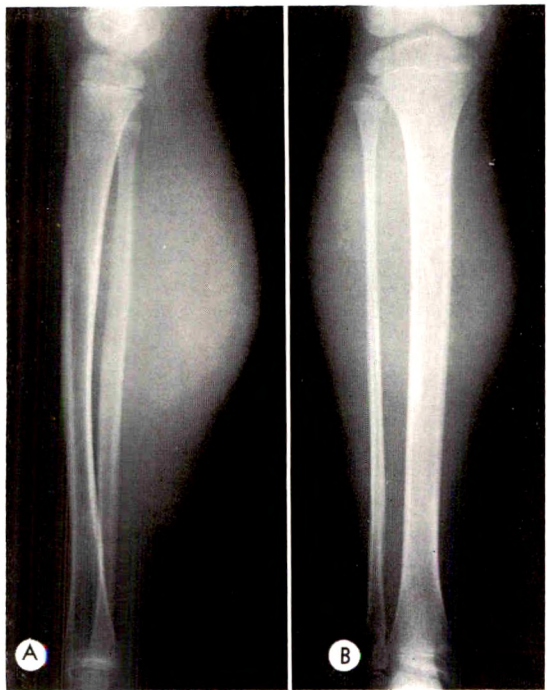


FIG. 1. (A) Lateral and (B) anteroposterior roentgenograms of the tibia and fibula in an 8 year old boy with Duchenne muscular dystrophy verified by a myopathic electromyogram. Symptoms began at the age of 1 year. The fibula is thickened in lateral view although normal in frontal view.

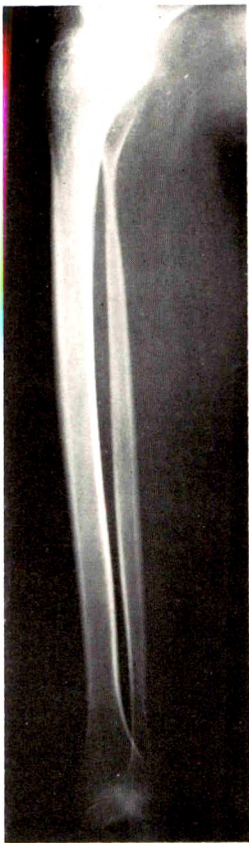


FIG. 2. Lateral view of tibia and fibula in a 17 year old boy with proven Duchenne muscular dystrophy. He is in a wheel chair. The fibula is thickened throughout most of its shaft.

For comparison, studies were also performed in 19 patients with the following neuromuscular disorders: Werdnig-Hoffmann disease, polymyositis, Charcot-Marie-Tooth disease, myotonia dystrophica, and hereditary proximal spinal muscular atrophy. Lateral roentgenograms of the tibia and fibula were also obtained in 51 subjects (ages 1-23 years) who were free of neurologic disease. These subjects were trauma cases and normal siblings of affected patients.

Lateral roentgenograms of the leg were used to determine the fibula/tibia (F/T) ratio, which was calculated as the widest anteroposterior diameter of the fibula divided by the smallest anteroposterior diameter of the tibia.



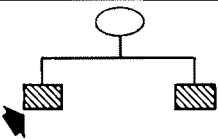
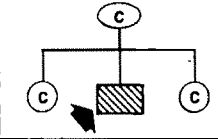
AGE OF PATIENT	FAMILY HISTORY	CPK	ABNORMAL EKG	EMG	F/T RATIO
9		19.6	+	Myopathic	.85
20		1.68	+	Myopathic	.74
10	Many Affected Males on Maternal Side	7.7	+	Myopathic	.98

FIG. 3. Findings in 3 patients with Duchenne muscular dystrophy. ■ = affected males. Arrow points to present case. ○ = females. ⊙ = carrier state determined by abnormal serum creatine phosphokinase levels. CPK = creatine phosphokinase; EKG = electrocardiogram; EMG = electromyogram; F/T ratio = fibula/tibia ratio.

#### RESULTS AND DISCUSSION

Twenty-five patients were diagnosed as having Duchenne muscular dystrophy on the basis of clinical course, myopathic electromyogram, elevated serum creatine phosphokinase and, in selected cases, muscle biopsies. Their ages ranged from 2 to 21 years. The patients with limb girdle and facioscapulohumeral dystrophies are considered separately.

The group with Duchenne dystrophy had an F/T ratio (mean  $\pm$  1 standard deviation) of  $0.77 \pm 0.15$  compared with  $0.59 \pm 0.11$  for the group with other neuromuscular disorders and  $0.58 \pm 0.07$  for the normal controls (Table II).

The widening of the fibula in lateral view involves most of the diaphysis. The tibia may be narrowed concomitantly, probably through atrophy. As a result, the widest diameter of the fibula in a lateral view may equal and occasionally exceed the narrowest diameter of the tibia. In the anteroposterior view the normal relationship of the tibia and fibula is preserved. Figure 1, A and B, demonstrates the thickened shaft of the fibula in an 8 year old boy with Duchenne muscular dystrophy. He is still able to walk but falls frequently and cannot dorsiflex his

foot. The anteroposterior roentgenogram is normal. Figure 2 is a lateral roentgenogram of the leg of a 17 year old boy. The diaphysis of the fibula is widened in relation to the tibia and, in addition, the neck of the fibula is narrowed. Figure 3 shows the data from 3 typical patients with proven Duchenne muscular dystrophy.

An F/T ratio above 0.70 is definitely abnormal, between 0.66 and 0.70 is borderline, and 0.65 or below is normal. Table III compares the F/T ratios in Duchenne mus-

TABLE III  
FIBULA/TIBIA (F/T) RATIO IN  
NEUROMUSCULAR DISEASE

F/T Ratio	No. of Patients	
	Duchenne Muscular Dystrophy	Other Neuromuscular Disorders
Abnormal $>0.70$	16	3
Borderline $0.66-0.70$	3	3
Normal $<0.66$	6	13
Total	25	19

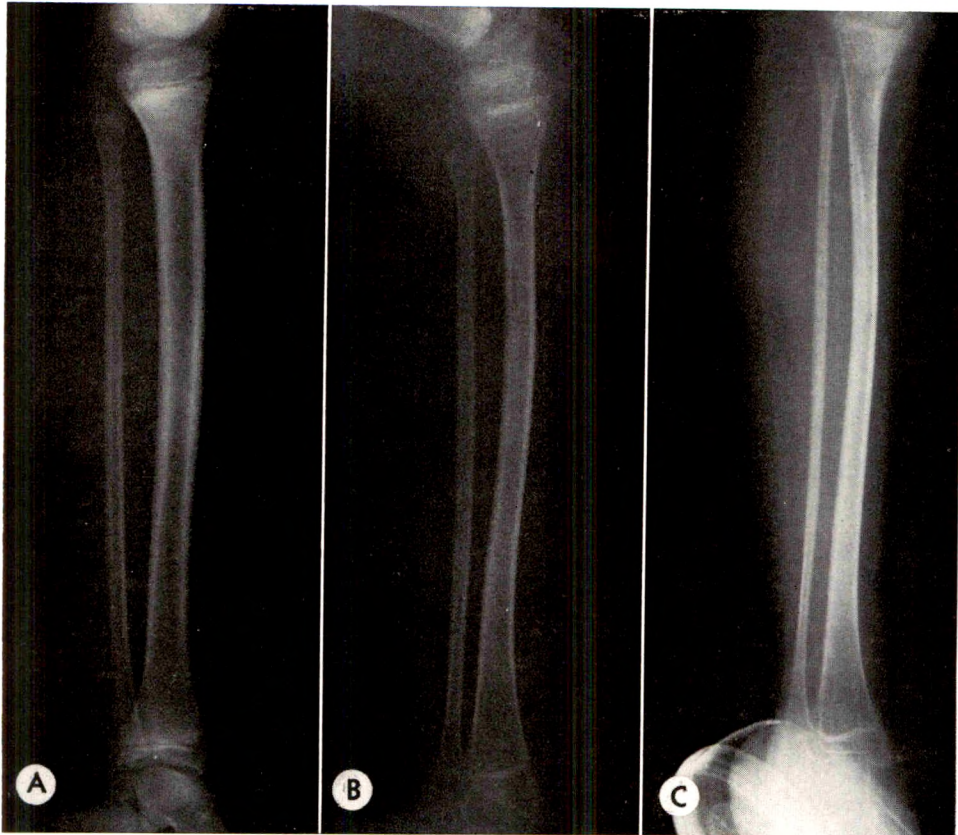


FIG. 4. (A-C) Lateral roentgenograms of the tibia and fibula. The 2 children in B and C, ages 12 and 13 years, are affected with hereditary proximal spinal atrophy. The roentgenogram in A is of their normal sibling, aged 9 years. The fibulae in the affected children are atrophied.

cular dystrophy with those in other neuromuscular disorders. The F/T ratio was abnormal in 19 of 25 patients with Duchenne muscular dystrophy, and in 3 of 4 with non-Duchenne muscular dystrophy. It was normal in 13 of 19 patients with other neuromuscular disease and in 49 of 51 controls.

Six patients with Duchenne muscular dystrophy had normal F/T ratios. One of these had far advanced disease. Four others were below the age of 5 years and the remaining patient was the only girl with Duchenne dystrophy, presumably of the autosomal recessive variety. The possibility that the F/T ratio is an age-related variable was tested, in part, as follows. The normal controls were divided by age into two groups. Twenty-five subjects were 1 to 12 years old, 26 were 13 to 23 years old. The F/T ratios of these two groups were not

significantly different by Student's *t* test.

In the 3 patients with limb girdle dystrophy, the F/T ratio was abnormal in 2 and borderline in 1. The one patient with facioscapulohumeral dystrophy had a normal F/T ratio despite pseudohypertrophy of the gastrocnemius muscles. These 4 patients walked not being severely affected.

The value of these measurements is demonstrated by the findings in a family affected with hereditary proximal spinal muscular atrophy<sup>13</sup> (Fig. 4, A, B and C). Although 4 children had muscle atrophy and contractures clinically indistinguishable from those seen in muscular dystrophy, their F/T ratios were normal. Neuropathic electromyograms and normal levels of serum creatine phosphokinase confirmed the diagnosis of neurogenic myopathy.

Muscular dystrophy patients tend to



have a thickening of the fibular shaft in an anteroposterior direction, while normal subjects and patients with other neuromuscular diseases do not. A reasonable, but unproven, pathogenetic factor may be the selectivity of muscle groups affected in early dystrophy.<sup>15</sup> The gastrocnemius is involved early while the dorsiflexors of the toes, which take origin from the fibula, are maintained until late in the disease. Possible evidence against this explanation is the frequent finding of a thickened fibula in early cases of dystrophy exhibiting only a small loss of muscle strength as well as in patients with far-advanced disease.

Thus, determination of the F/T ratio appears to be of definite value in the diagnostic work-up for Duchenne muscular dystrophy.

#### SUMMARY

1. A frequent finding in Duchenne muscular dystrophy is increased thickness of the fibula in an anteroposterior direction, usually involving most of the diaphysis.

2. The relationship between the fibula and tibia may be expressed as the fibula/tibia (F/T) ratio. An F/T ratio above 0.70 is abnormal.

3. The fibula is normal in children with Duchenne muscular dystrophy who are under the age of 5 years.

4. The F/T ratio in limb girdle dystrophy often is also abnormally large.

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## UPRIGHT PLANIGRAMS IN PNEUMO- ENCEPHALOGRAPHY

By GOMER W. ROBERTS, M.D.  
FORT WORTH, TEXAS

IN THE brilliant textbook "Diagnostic Neuroradiology," by Taveras and Wood, many challenging suggestions are presented for the conduct of a pneumoencephalographic procedure. It is suggested that detail of the anatomy of the posterior fossa is best demonstrated early in the study and that filling of these structures is usually best shown if roentgenograms can be obtained with the patient in the upright position. It is also suggested that if planigraphic equipment is available it should produce clearer definition of midline structures.

Most x-ray departments have planigraphic equipment, but its adaptation to upright work is usually either not possible or difficult at best. Further, in order to use conventional planigraphy with the patient in some form of upright support for pneumoencephalography, there is a substantial distance between the mid-line of the skull and the plane of the film within the Bucky tray. A patient undergoing pneumoencephalography usually is not able to co-operate fully and substantial stabilizing equipment is necessary to immobilize the patient during planigraphy. A patient secured in a somersault pneumoencephalographic chair in upright position presents an ideal opportunity for planigraphy during early filling. An attempt was made to construct a simple planigraphic attachment specifically to work with a conventional ceiling-mounted roentgenographic tube and a somersault pneumoencephalographic chair. This simple machine is the subject of this report.

### DESIGN AND CONSTRUCTION

The four elements of the manually-operated planigraphic device are shown in Figure 1. These components are not precision pieces of equipment. They could be

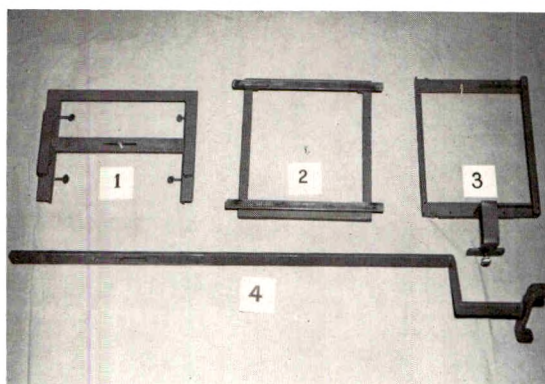


FIG. 1. Components of the upright planigram attachment: (1) Fulcrum support plate; (2) cassette carrier track; (3) cassette carrier; and (4) planigraphic arm.

fabricated in a moderately well equipped home work shop where the most sophisticated equipment needed would be a drill press and an arc welder. The only operation requiring skilled machining is the cutting of the slots for the fulcrum bearing. This operation can be subcontracted to any machine shop at a token fee.

The fulcrum slots were cut so that planigrams lateral to the mid-line are possible. The slots can be extended as far as desired so that upright planigrams may be obtained at any level throughout the entire skull.

### OPERATION

The fulcrum support plate is placed in the rear of the head holder device and fastened with the thumb screws. This fulcrum support and the planigraphic arm usually are not disassembled and are removed from the machine as a single unit. The cassette carrier and the cassette carrier track are left coupled together. These two pieces are slipped into the head holder in the slots ordinarily occupied by a cassette during routine roentgenography. The base plate of the cassette carrier track is built



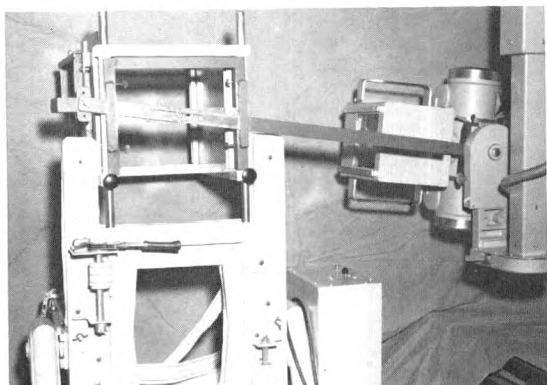


FIG. 2. Machine assembled and installed on head-supporting device of somersault pneumoencephalographic chair.

to the same dimensions as a 10×12 inch cassette. It is perhaps even better if it is slightly oversized so that it requires a mild degree of force to insert it. This would prevent any undesired movement of the track during the planigraphy.

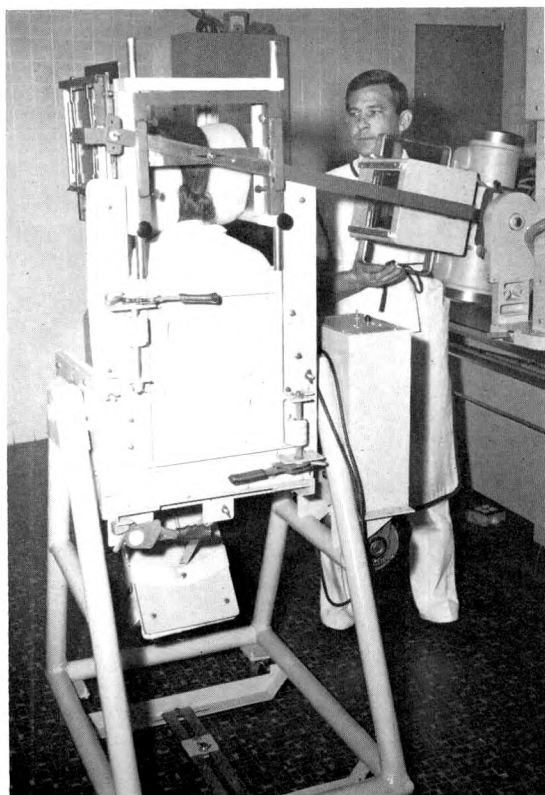


FIG. 3. Planigraphic machine in operation (Student X-ray Technician in the chair as a model).

arm is coupled to the cassette carrier by the single thumb screw. In our particular operation two additional thumb screws are used to fasten the planigraphic arm to the ceiling-mounted roentgenographic tube housing. The completed assembly is shown in Figure 2. A stationary grid is used and it is oriented with the center line of the grid in a vertical direction. This allows the central ray to follow the center line of the grid during the planigraphy.

After the injection of 10 to 20 cc. of air into the lumbar subarachnoid space, a planigraphic cut is made. More are made as small increments of air are added at the discretion of the neurosurgeon. The x-ray technician moves the ceiling mounted tube manually through an arc which measures between 3 and 4 inches (Figure 3). Arcs of greater length than this have tended to produce considerable blurring of the detail of the mid-line structures, as well as more lateral structures. Good clean detail is achieved with even a small arc of 3 inches tube travel. Although many techniques would work, we have used about 2 seconds



FIG. 4. Mid-line planigram showing good detail of the mid-line ventricular anatomy.

to complete the arc with about 15 ma. at 74 kv. for a normal skull. With only a few minutes practice, an x-ray technician can carry the tube through a smooth arc so that the tube travels the proper distance within the desired time, in our case 2 seconds.

#### DISCUSSION

The planigrams obtained with the use of the manually operated planigraphic device have usually shown excellent detail of the fourth ventricle, aqueduct, and posterior third ventricle (Fig. 4). Filling is routinely done with the head well flexed. This usually results in a fluid level obliquely across the third ventricle, but with good display of the anatomy above and posterior to the level of the massa intermedia (Fig. 5). There usually is some gas which reaches the pontine cistern and the cisterna ambiens. The gas in the upper cervical spinal canal and the cisterna magna also is well demonstrated on the mid-line planigrams.



FIG. 5. Mid-line planigram showing fairly good detail of mid-line anatomy, especially the posterior third ventricle. Note the well developed supra-pineal recess.

There is a slightly greater distance from the mid-line of the skull to the film than in routine roentgenographic table work. It is expected that a slight degree of magnification results. It may be necessary in our cases to slightly revise the range of normal measurements for the position of the fourth ventricle and aqueduct because of this slight magnification.

It is desirable and at times extremely valuable to have an occipital or half axial projection of the skull early in the pneumoencephalographic study (*i.e.*, while there is still gas in the fourth ventricle, aqueduct and third ventricle). For this reason, the fulcrum support was constructed with no bottom cross member. This allows a film to be exposed without bothering to remove the planigraphic equipment from the head holding device. In our case we have used a portable roentgenographic machine rather than to uncouple the ceiling tube from the planigraphic arm. This allows planigrams and half-axial studies to be obtained very early in the procedure to visualize the posterior fossa structures when they are optimally filled.

While there is some thrust against the chair by the movement of the roentgenographic tube in its ceiling carrier, the chair is sufficiently heavy, especially when a patient is in it, that it does not move during the planigraphic procedure.

It is quite probable that a similar planigraphic device could be fabricated to fit any roentgenographic equipment. In all probability, a similar device could be fabricated to fit any of the available somersault pneumoencephalography chairs also. There are modifications or alterations of this device which have been suggested and which do have some merit. It would be desirable if planigrams could be cut with the patient supine or prone while in the pneumoencephalographic chair. Either of these last two positions could be technically accomplished fairly easily by minor variation of the means of attaching the planigraphic arm to the tube. It would seem quite worthwhile to have a means to cut planigrams



with the patient supine and, in this manner, the anatomy of the anterior third ventricle could be portrayed most clearly. Working with the ceiling-mounted tube, a universal bracket has not been possible and it will be necessary to fabricate two different means of coupling the planigraphic arm to the ceiling-mounted tube in order to accomplish these two completely different types of movement of the tube, one vertical and the other horizontal. Perhaps the simplest of all might be a coupling arrangement whereby the cone of the roentgenographic tube is coupled to the fulcrum arm using the slots on the face of the cone. A square shaped bracket could be fabricated so that this could be inserted in the cone for horizontal or vertical type movement.

It would be possible to build an electric motor drive for the planigraphic attachment, but doing this would involve a rather substantial increase in cost, as well as making the instrument considerably more bulky and awkward to handle. The present device can be removed from the pneumoencephalographic chair in less than 1 minute and routine pneumoencephalographic filming can proceed without undue delay.

The amount of radiation received by the personnel operating the equipment would not be expected to be as high as that received when directly performing autotomography and since this ordinarily involves only 2 or 3 roentgenograms made with the technician wearing a lead apron, this radiation is not considered a significant exposure to technical personnel. At no time is the technician exposed to the direct beam from the machine.

#### SUMMARY

A manually operated planigraphic device is described for use with a somersault pneumoencephalographic chair. This device is simple in design and could be fabricated to fit most upright pneumoencephalographic equipment and adjusted to most roentgenographic tubes.

Planigrams obtained with this device show excellent detail of the mid-line ventricular structures. The procedure has been most valuable when used during the early filling phase of pneumoencephalography.

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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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## EDITORIALS

### THE RADIOLOGIST'S RESPONSIBILITY FOR NEURORADIOLOGY

THE continuing advancement of interest in neuroradiology as evidenced by the publication of many related articles, such as those in this present issue, invites radiologists to consider their responsibilities in this field. In many instances these will be shared depending upon the training and experience of the physicians involved as well as upon the organizational structure of the institutions in which persons with neurologic diseases are cared for. Examinations of these patients should be designed to provide the most useful information for the subsequent management of their problems consistent with the least risk of morbidity or mortality.

Before the advent of the so-called special procedures which require certain operative techniques, nearly all complex roentgenographic examinations were carried out in an x-ray department under the supervision of a trained, interested radiologist. Procedures requiring introduction of needles or catheters were initiated and to a considerable extent developed by physicians more concerned with surgical disciplines than with the formal application of the roentgen ray to diagnosis. Enhancement and refinements of these examinations now, however, require the proper use of expensive, complicated, and sophisticated roentgenographic equipment in addition to the expert operative manipulation of apparatus for the introduction of foreign materials such as opaque contrast solutions and various gases. Although in some situations a certain rivalry has grown up between radiologists and other specialists relative to the responsibility for these procedures, it would seem that such examinations are sufficiently complex to require the best efforts of

several related disciplines in order to provide a maximum in service to the patient.

Persons who are likely to undergo these difficult and complicated procedures usually should be evaluated by a competent neurologist, and thereafter the choice of the most rewarding and least hazardous of these special studies may be discussed by the neurologist, radiologist, and neurosurgeon. Each should be permitted to present his point of view tactfully and courteously. Because in many instances these examinations disclose the necessity of early neurosurgical intervention, it is reasonable to leave the final choice of the procedure required with the neurosurgeon especially in the rare situation in which there is an unresolved difference of opinion among these three specialists.

Responsibility for the clinical evaluation of the patient prior to such examinations frequently belongs to the medical neurologist although following the roentgenologic procedures the neurosurgeon is usually in charge. During the time the patient is in the radiology department for a specific procedure, the radiologist should be primarily responsible. This, of course, is true only if the radiologist is well trained and competent in all the aspects of the examination to be undertaken.

These procedures are a combination of three different but related functions: operative, technical, and diagnostic. One should not consider himself competent in the performance of these examinations unless he is accomplished in all three phases. The introduction of a needle or a catheter into a vessel is no more the performance of an angiographic examination than is the isolated interpretation of the films or the

proper selection of roentgenographic technical factors. In many hospitals at this time there is unfortunately no one physician who has been completely trained in these three aspects, and therefore it is appropriate that particular segments of the examination be undertaken by those best qualified in the specific features involved. At the same time, unless unusual circumstances supervene, one physician, usually the radiologist, should be in charge so that the examination in his department can be carried out smoothly and safely with excellent diagnostic results. This does not mean that one individual must necessarily perform all of each procedure in every instance although he should be intimately involved in each examination under his direction and be able to assist in solving any difficulties that may arise. Only in this manner it is possible for several different diagnostic studies to proceed simultaneously within the same department.

Once the examination has been completed and the diagnosis established through a mutual understanding between the neuroradiologist, neurologist, and neurosurgeon, under most circumstances the patient should be under the care of the neurosurgeon, either because of required operative intervention or for observation following the examination to check for any possible latent complications. Under some conditions it may be more appropriate for him to be under the supervision of the medical neurologist.

The radiologist's responsibility does not end with his activities concerning the choice, performance, and analysis of these examinations. There are some hospitals in this country in which there is no neuroradiologic service available. There may, however, be a neurosurgeon and it is important for him to be able to perform the required test with all the help he can obtain from the radiologist in charge of the hospital department. Therefore, in institutions in which neurosurgeons are being trained, it is important that the neuroradiologist give freely of his time and efforts to supplement

the instruction of young neurosurgeons in the various aspects of neuroradiology. In busy medical centers in which large numbers of neuroradiologic procedures are undertaken, there probably would not be any difficulty in providing a sufficient amount of experience for the proper training of neuroradiologists and neurosurgeons. It would also seem to be desirable for the neurologist to understand these examinations and their interpretation in order to better evaluate his patient. However, it does not seem necessary to complicate the neurologist's already busy practice with the specific performance of these tests, particularly because neurosurgical procedures may be required soon after such an examination is completed and there might be an unfortunate delay if the neurosurgeon were not already involved.

It is imperative, however, that in those institutions in which young physicians are trained in all phases of the diagnosis and care of patients who have neurologic diseases, there be one or more radiologists completely trained and highly skilled in neuroradiology. They should not only supervise the performance of the special procedures but also train young radiologists for careers in this field. There should also be instruction for neurosurgeons and general radiologists in many aspects of these procedures. This need was recognized several years ago by many groups including the National Institute of Neurological Diseases and Blindness and therefore there are now a small number of institutions in which formal programs for the training of neuroradiologists are available. The candidates are selected from among those who have completed the academic requirements for certification by the American or Canadian Board of Radiology and who are willing to spend 2 or more additional years to obtain this specialized neuroradiologic training. This includes instruction in the basic neurologic sciences sufficient for an understanding of the clinical problems presented and for assisting in the selection of the most appropriate examination to solve





GEORGE W. GRIER, M.D.

1882-1966

AFTER a lingering illness, Doctor Grier died in Pittsburgh, his adopted city, on July 23, 1966. He had been born and raised in Sewell, New Jersey, where he maintained a country home to which he returned frequently. His medical education was received at Jefferson Medical College from which he graduated in 1904.

He was proud of being named for the father of this country and would tell someone now and then that his initials stood for George Washington. A mild-mannered, likeable man, without pretension, he lived through the Era of the Roentgen Pioneers and the Golden Age of Radiology, to the beginning of the Atomic Phase in the medical

application of *The Invisible Light*.\*

Doctor Grier came to Pittsburgh in 1904 as an intern at the West Penn Hospital at which time the practice of Radiology was extremely primitive. In fact, he told me the only x-ray work being done at the hospital then was fluoroscopy, and this only in the erect position since the tilt table had not been invented. Two years later he took over the X-Ray Department at the West Penn Hospital in Pittsburgh, and then obtained one of the earliest types of mica disk static machines with an output of approximately 37 kv. at 4 ma., so that it took approximately 4 minutes to make an x-ray examination of the pelvis. At that time Doctor Grier was following the usual practice of using a fluoroscope to look at his hand while the tube was operating to test the penetrating quality of the beam. Since some people had already received serious x-ray injuries from such use, he promptly dropped this procedure and said that he had not kept it up for longer than a year. It is a testament to his acute discernment at this early date in Radiology that he had practically no evidence of injury from x rays or radium throughout a period of 60 years of use. He always emphasized the importance of protection from ionizing radiation to all whom he taught.

As was common in the early days, he promptly became a peripatetic radiologist. In 1908 he opened an x-ray office in downtown Pittsburgh and had been appointed radiologist on the staff of St. Margaret's Hospital in Pittsburgh. (At various times during his career, Doctor Grier was either on the staff as a consultant or radiologist at 16 different hospitals.)

In an interview I had with Doctor Grier in May 1960,† he told me that a radiologist on the staff of a hospital in those days was in the same capacity as that of any other specialist who, when a person needed an

x-ray examination in the hospital, was seen by the radiologist who brought his equipment to the hospital and made his examination there, took the glass plates back to his office, processed them and made a report. For this he sent his bill to the patient, the same procedure as any other specialist who had been called in for a consultation.

Doctor Grier told me that this went on for some time until about the beginning of the First World War. At its end radiologists who had been trained by the Army School of Roentgenology did not want to go into private office practice of Radiology so it was not easy for them to carry on as mentioned. In this way, hospitals developed the system of supplying the equipment and hiring these younger radiologists with limited experience on a salary and collecting a large part of the fees for themselves.

Doctor George C. Johnston had started x-ray work in 1902 and had been the radiologist at Allegheny General Hospital, St. John's Hospital, Presbyterian Hospital as well as Passavant, St. Joseph's and Montefiore Hospitals in Pittsburgh. When they began their partnership in 1913, Doctor Johnston was 10 years older than Doctor Grier and was the first Professor of Radiology at what was then the Western University of Pennsylvania (WUP). At this time Doctor Johnston appointed his Associate, Doctor Grier, an Instructor. In the meantime, Doctor Grier did all the x-ray work at the hospitals where Doctor Johnston was on the staff. He also took care of their office work during the time when Doctor Johnston served in the First World War from which he returned in the fall of 1919. A year later, Doctor Johnston had a heart attack, was incapacitated and retired. It was then that Doctor Grier became Assistant Professor of Radiology at the new University of Pittsburgh and rose through the ranks until 1933 when he was appointed full Professor of Radiology and Chairman of the Department—a position which he held until 1953 when he became Emeritus Professor of Radiology.

Doctor Grier will be remembered by

\* *THE TRAIL OF THE INVISIBLE LIGHT*, Grigg, 1965, Charles C Thomas, Publisher, Springfield, Illinois. See Grier: pp. 202, 203, 227, 228, 234, 273.

† Tape Recording at a meeting of the Pittsburgh Roentgen Society, May 29, 1960, devoted to *Early History of Radiology in Pittsburgh*.



many in Pittsburgh as having been our undergraduate teacher of Radiology, particularly Roentgen Diagnosis, then our preceptor as Residents in the University of Pittsburgh Medical Center and during his later years there as a full Professor.

Throughout the years, he had been active in medical societies and had been a member of the Board of Directors of the Allegheny County Medical Society from 1921 through 1934 (President 1933-1934), served as Secretary of the Section on Radiology of the American Medical Association from 1927 through 1932. From 1917 to 1920 he was Secretary of the American Roentgen Ray Society and became its President in 1934-1935. He was also Secretary of the American Radium Society from 1927 to 1931 (attesting to his long continued inter-

est in radiation therapy) and President of that Society in 1935-1936. In addition to being honored with election to these high offices, he was a member of the Board of Chancellors of the American College of Radiology from 1947 to 1951.

He is survived by his wife, the former Ida Hammer of Pittsburgh, and three grandchildren.

Doctor Grier left a heritage of high professional standards and ethics, as well as a fine tradition of paramount regard for his patients' interest, to all who had the pleasure of being his friends and academic professional descendants.

LEWIS E. ETTER, M.D.

Pinewood Farm  
Warrendale, Pennsylvania 15086



## NEWS ITEMS

### ELEVENTH ANNUAL CLINICAL CONFERENCE

M. D. ANDERSON HOSPITAL AND  
TUMOR INSTITUTE

"Cancer of the Uterus and Ovary" will be the subject of the Eleventh Annual Clinical Conference to be held at The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas, on December 1 and 2, 1966.

Outstanding physicians from institutions throughout the United States and one from Sweden will join staff members from The University of Texas M. D. Anderson Hospital and Tumor Institute in presenting the most current information on diagnostic procedures and treatment for carcinoma of the uterus, uterine cervix, and ovary.

Co-sponsor of the conference with M. D. Anderson Hospital is the Division of Continuing Education of The University of Texas Graduate School of Biomedical Sciences at Houston.

Further information concerning the conference may be obtained by writing to Dr. Felix Rutledge, Chairman, 1966 Clinical Conference, The University of Texas M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, Texas 77025.

### NINETEENTH ANNUAL MIDWINTER RADIOLOGICAL CONFERENCE

The Nineteenth Annual Midwinter Radiological Conference, sponsored by the Los Angeles Radiological Society will be held at the International Hotel (adjacent Los Angeles International Airport), Los Angeles, California, on Saturday, January 28 and Sunday, January 29, 1967.

The program will include the following speakers: G. J. D'Angio, M.D., University of Minnesota, Minneapolis, Minnesota; Benjamin Felson, M.D., University of Cincinnati, Cincinnati, Ohio; Dr. Herbert J. Kaufmann, Basler Kinderspital, Basel,

Switzerland; Dr. John W. Laws, Radiodiagnostic Hospital, Hammersmith Hospital, London, England; and Richard Lester, M.D., Duke University, Durham, North Carolina.

Hotel reservations may be made by contacting the Convention Manager, International Hotel, Sepulveda at Century, Los Angeles, California.

Conference reservations should be made through Richard R. Scriber, M.D., 2400 South Flower Street, Los Angeles, California 90007.

For further information write to Calvin Williams, M.D., Publicity Chairman, Midwinter Radiological Conference, 15107 Vanowen Street, Van Nuys, California 91405.

### TENTH ANNUAL POSTGRADUATE COURSE

UNIVERSITY OF CALIFORNIA SAN  
FRANCISCO MEDICAL CENTER

The Tenth Annual Postgraduate Course in Diagnostic Radiology to be presented by the Department of Radiology and Continuing Education in Medicine and the Health Sciences of the University of California San Francisco Medical Center will be held March 8-12, 1967.

It is an intensive five-day course which includes lectures, panel discussions, symposia, exhibits, and a social hour. Registrants may enroll for the full five days or by the day.

The Program Chairman is Alexander R. Margulis, M.D., Professor and Chairman, Department of Radiology, University of California School of Medicine, San Francisco; the Program Director is Howard L. Steinbach, M.D., Professor of Radiology; and the Assistant Program Director is Charles D. Noonan, M.D., Assistant Professor of Radiology, University of California, School of Medicine, San Francisco, California.



The course will be conducted by the faculty of the Department of Radiology of the University of California School of Medicine, San Francisco, and the following guest speakers: Walter E. Berdon, M.D., New York, New York; Milton Elkin, M.D., New York, New York; Robert H. Freiburger, M.D., New York, New York; Mordecai Halpern, M.D., Los Angeles, California; William Hanafée, M.D., Los Angeles, California; John A. Kirkpatrick, Jr., M.D., Philadelphia, Pennsylvania; George Simon, M.D., F.F.R., London, England; Manuel Viamonte, Jr., M.D., Miami, Florida; and Jerome F. Wiot, M.D., Cincinnati, Ohio.

For further information and application for enrollment contact Seymour M. Farber, M.D., Dean of Educational Services and Director of Continuing Education, Health Sciences, University of California Medical Center, San Francisco, California 94122.

VII CONGRESO DE ELECTORADIOLOGOS  
DE CULTURA LATINA  
AND  
I CONGRESO DE LA ASOCIACIÓN  
EUROPEA DE RADIOLOGÍA

The Seventh Congress of Electroradiologists of Latin Culture joined by the First Congress of the European Association of Radiologists will take place April 2-8, 1967 in Barcelona, Spain, under the Honorary Presidency of Prof. G. F. Leroux and Prof. B. Rajewsky.

The officiating President is Dr. F. Galvez of Madrid, and the Secretary General, Dr. F. Manchón Azcona of Barcelona.

A very elaborate scientific program is being arranged encompassing the branches of Radiodiagnosis, Radiotherapy, Radiobiology, Techniques, Physics and Education in Radiology.

The many Social Activities will be in the time-honored tradition characteristic of Spain.

For further information please write to Secretary General, Dr. F. Manchón Azcona, Facultad de Medicina, Barcelona, Spain.

SOCIÉTÉ EUROPÉENNE DE  
RADIOLOGIE PÉDIATRIQUE

The 1967 Annual Meeting of the Société Européenne de Radiologie Pédiatrique will be held April 13-15 at Basel, Switzerland.

The officers for 1966-67 are: *President*, Dr. H. J. Kaufmann; *Secretary-General*, Dr. H. Ludin, Department of Roentgenology, Basler Kinderspital, Basel, Switzerland.

The *Permanent Secretary* is Dr. Jaques Sauvegrain, Hôpital des Enfants Malades, 149, rue de Sèvres, Paris 15e, France.

For further information contact Dr. H. J. Kaufmann, Leiter des Röntgeninstituts, Basler Kinderspital, 4000 Basel 5, Switzerland.

THE BRITISH INSTITUTE OF RADIOLOGY

The Twenty-eighth Annual Congress with technical and scientific exhibitions will be held April 26-28, 1967 at the Central Hall and Caxton Hall, Westminster, London, S.W. 1.

The Scientific Program will include:

*Joint Session.* The Mackenzie Davidson Memorial Lecture: Computers in Radiology. *Diagnostic Radiology.* Disobliteration of Femoral Arteries. Investigations of the Pancreas.

*Radiotherapy.* Long-Term Follow-Up Studies in Radiotherapy. Proffered Papers on Radiotherapy and Radiological Physics.

*Joint Meeting of Isotope Study Section and Radiobiology Sections.* Selective Uptake of Radio-Isotopes: Studies of Mechanism, Dosimetry and Applications in Diagnosis and Therapy.

It is hoped to include a session on Apparatus and Technology and that there will also be parallel sessions for Radiobiologists.

Technical Exhibition will be staged by the British X-Ray Industry on 2 floors of the Central Hall. The last exhibition was held in April, 1964, since then there has been considerable technical progress. In addition to British made equipment, imported products will be shown by established agents of overseas manufacturers.

For provisional program and further

information, please write to The British Institute of Radiology, 32 Welbeck Street, London, W. 1, England.

#### NINTH INTER-AMERICAN COLLEGE OF RADIOLOGY

The date chosen for the Meeting of the Ninth Inter-American College of Radiology is December 6-12, 1967; and the headquarters of the meeting will be the Hotel San Rafael de Punta del Este, Uruguay.

The themes chosen for the Ninth Congress of Radiology are the following:

##### *Radiodiagnosis*

- (1) Radiodiagnosis of the Cerebral Circulatory Insufficiency
- (2) Radiodiagnosis of Acquired Cardiopathies
- (3) Radiodiagnosis of Congenital Bone Dystrophies
- (4) Mammography
- (5) Bone Microradiography

##### *Radiotherapy*

- (1) Fractionation
- (2) Hybaroxic Radiotherapy
- (3) Radiotherapy of Tumors of the Hypopharynx

##### *Nuclear Medicine*

Diagnosis of Physical Abnormalities and

Dysfunction of the Kidney, Liver and Brain

In addition to the above, the Congress will be open to free themes.

Those interested please write to the Secretaries: Dr. Helmut Kasdorf and Dr. Roberto Francois, Cassilla de Correo No. 930, Montevideo, Uruguay.

The Counselor for the United States to the Inter-American College of Radiology is Juan A. del Regato, M.D., Penrose Cancer Hospital, 2215 North Cascade Avenue, Colorado Springs, Colorado 80907.

#### DEPARTMENT OF RADIOLOGY TEMPLE UNIVERSITY CENTER FOR THE HEALTH SCIENCES

##### 1966 W. EDWARD CHAMBERLAIN LECTURE

The 1966 W. Edward Chamberlain Lecture will be given by Borje Rudewald, M.D., Docent in Medical Techniques, Stockholm, Sweden, on Wednesday, November 23, 1966 at 4:00 P.M.

The title of the lecture is: "Blood Flow in the Human Aorta—Radiologic and Physiologic Analysis," and will be held in the Auditorium—3rd floor, Temple University School of Medicine, Broad Street at Ontario, Philadelphia, Pennsylvania.





## BOOK REVIEW AND BOOKS RECEIVED

*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

**PNEUMOENCEPHALOGRAPHY AND CEREBRAL ANGIOGRAPHY.** By Bernard S. Epstein, M.D., Clinical Professor of Radiology, The Albert Einstein College of Medicine, Yeshiva University, New York City; Director of Radiology, The Long Island Jewish Hospital, New Hyde Park, N. Y.; Radiologist-in-Chief, The Long Island Jewish Hospital-Queens Hospital Center Affiliation, Queens, N. Y. Cloth. Pp. 349, with many illustrations. Price, \$20.00. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill., 1966.

The ability to produce a textbook on pneumoencephalography and cerebral arteriography depends upon adequate knowledge of anatomy and sound practical experience. There is no doubt that Dr. Epstein possesses both of these requirements and this is reflected in his textbook.

The book is divided into 4 sections: Normal pneumoencephalogram and angiogram; Congenital malformations; Vascular malformations and intracranial hemorrhage; and Brain tumors. The description of the normal pneumoencephalogram is covered in detail and includes an excellent correlation with the anatomy of the intracranial structure. The illustrations are of good quality but suffer from a surfeit of labels in which the object is indicated by initials or letters. This causes difficulty in interpretation when the reader is forced to oscillate between rather detailed and lengthy legends and the illustration. This section might have been improved by the use of line drawings next to the illustrations showing the salient features in semidiagrammatic form. The labeling is less of a problem in the section on normal angiography and the illustrations are again of good quality.

There could have been more detailed anatomic description in this section, which is not up to the standard of that on the normal pneumoencephalogram. Both pneumoencephalography and arteriography would have benefited by a discussion on technique. One wonders how Dr. Epstein obtains his beautiful pneumoencephalograms. What is his secret?

The rest of the book consists of an excellent presentation of the various subjects. One chapter on brain tumor is particularly good with the term "tumor" used in its widest sense to include granulomas and brain abscesses. There is no doubt that this chapter alone would be sufficient recommendation to the reader interested in pneumoencephalography or arteriography.

There is one omission which could be rectified in subsequent editions. One of the major uses of arteriography is in the diagnosis and treatment of cerebrovascular disease and a chapter on this subject would, I think, complete a useful and well written textbook.

JOHN GILROY, M.D., F.R.C.P. (C).

### BOOKS RECEIVED

**PROGRESS IN CLINICAL CANCER.** Edited by Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Attending Surgeon, New York Medical College, Flower and Fifth Avenue Hospitals; Attending Surgeon and Chief of the Soft Somatic Tissue Tumor Service, Hospital for Joint Diseases; Attending Surgeon, Pack Medical Group, New York, N. Y. Volume II. Cloth. Pp. 375, with some illustrations. Price, \$15.75. Grune & Stratton, Inc., 381 Park Avenue South, New York, N. Y., 1966.

**DIE WIRBELSÄULENLEIDEN UND IHRE DIFFERENTIALDIAGNOSE.** By Prof. Dr. J. E. W. Brocher, Genf. Cloth. Pp. 684, with 511 illustrations. Price, Ganzleinen DM 166.-. Georg Thieme Verlag, Stuttgart. In U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y., 1966.

**RECENT ADVANCES IN NUCLEAR MEDICINE.** Based on a Symposium Sponsored by the Department of Radiology of the Hahnemann Medical College. Edited by Millard N. Croll, M.D., Associate Professor of Radiology, The Hahnemann Medical College, Philadelphia, Pa.; and Luther W. Brady, M.D., Professor of Radiology, The Hahnemann Medical College, Philadelphia, Pa. Cloth. Pp. 260, with many illustrations. Price, \$12.50. Appleton-Century-Crofts, 440 Park Avenue South, New York, N. Y., 1966.

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ABSTRACTS OF RADIOLOGICAL  
LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS	
NERVOUS SYSTEM	
ROSENBERG, J. C.: Arteriographic demonstration of compression syndromes of the thoracic outlet.....	766
STOLL, B. A., and ANDREWS, J. T.: Radiation-induced peripheral neuropathy.....	766
SANTÍN, G., and VARGAS S. J.: Roentgen study of cysticercosis of central nervous system....	766
SKELETAL SYSTEM	
GOLDBLOOM, R. B. <i>et al.</i> : Idiopathic periosteal hyperostosis with dysproteinemia: a new clinical entity.....	767
MORGAN, T., and STEPHEN, D.: Bone changes in urticaria pigmentosa: a report of a case...	767
MACHTEY, I., RODNAN, G. P., and BENEDEK, T. G.: Paget's disease of the hip joint.....	767
LANGLEY, G. R., SABEAN, H. B., and SORGER, K.: Sclerotic lesions of bone in myeloma....	768
BLOOD AND LYMPH SYSTEM	
BRENNER, H., and ZAUNBAUER, W.: Possibilities for error in cerebral angiography.....	768
VOGELSANG, H.: Possibilities for incorrect diagnosis of brain abscess by means of carotid angiography.....	769
MAURER, H.-J., FRIELINGSDORF, D., and VLEUGELS, E.: Possibilities for incorrect diagnosis in splenoportography.....	769
RÜTTIMANN, A.: Errors in lymphography.....	769
BRYANT, L. R., and SPENCER, F. C.: Occlusive disease of subclavian artery.....	770
VIGNE, J. <i>et al.</i> : The Pegot-Cruveilhier-Baumgarten syndrome.....	770
FONKALSRUD, E. W., LINDE, L. M., and LONGMIRE, W. P., JR.: Portal hypertension from idiopathic superior vena caval obstruction ..	770
DOTTER, C. T. <i>et al.</i> : The "nonsurgical" treatment of iliofemoral arteriosclerotic obstruction.....	771
JACKSON, R. J. A.: Complications of lymphography.....	771
DOLAN, P. A.: Lymphography: complications encountered in 522 examinations.....	772
GENERAL	
SALOMONI, E.: Turano's method applied to the study of motor-functions of hollow muscular viscera (simultaneous roentgen cinematographic and pressure recording).....	772
KOSLOWSKI, L., and WELLER, S.: The application of x-ray image intensification to orthopaedic surgery.....	773
WISE, R. E., and GANSON, J.: Subtraction technic: video and color methods.....	773
TEN CATE, H. W., JONGMANS, H., and ZIEDSES DES PLANTES, B. G.: Subtraction technique in renal angiography.....	773
RADIATION THERAPY	
KAUFMAN, B. <i>et al.</i> : Transnasal-transsphenoidal yttrium-90 pituitary implantation in the therapy of acromegaly.....	774
SANDLER, H. C.: Errors of oral cytodiagnosis: report of follow-up of 1,801 patients.....	774



## ROENTGEN DIAGNOSIS

## NERVOUS SYSTEM

ROSENBERG, J. C. Arteriographic demonstration of compression syndromes of the thoracic outlet. *South. M. J.*, April, 1966, 59, 400-403. (From: Department of Surgery, Maumee Valley Hospital, Toledo, Ohio.)

Neurovascular compression syndromes at the thoracic outlet were studied in 15 patients by arteriography. The femoral artery was punctured percutaneously in each case, and a catheter was positioned with its tip adjacent to the orifice of the subclavian artery. Serial arteriography was performed with the arm in the symptom-producing position.

The most commonly encountered mechanism of compression in this series was the "costoclavicular syndrome." With the shoulders thrown back in the military position of "attention," or with the arms hyperabducted, the clavicle moves backward and may constrict the costoclavicular space and thus the subclavian artery passing over the first rib through this space. The author recommends resection of the first rib and states that 3 patients so treated all obtained relief of symptoms.

The next most common mechanism in this series is a cervical rib. One case is reported showing compression of the subclavian artery and post-stenotic dilatation. With these findings, resection of the cervical rib is indicated.

The scalenus anticus syndrome was demonstrated in only 1 patient in this series. This is probably a far less common cause of thoracic outlet compression than was once thought, which might explain the high failure rates reported with scalenectomy.

The author states in his conclusions that arteriography should not be necessary for diagnosis. However, it is evident from this report and the experience of others that it is only by arteriography in the symptom-producing position that accurate diagnosis can be made and the proper therapeutic procedure selected.—*Stephen A. Kieffer, M.D.*

STOLL, BASIL A., and ANDREWS, JOHN T. Radiation-induced peripheral neuropathy. *Brit. M. J.*, April 2, 1966, 1, 834-837. (From: Peter MacCallum Clinic, Melbourne, Australia.)

In rebuttal to the long held opinion that the peripheral nerves of adults exhibit a remarkable degree of resistance to ionizing radiation, the authors present evidence of peripheral nerve damage in a group of patients treated with megavoltage roentgen therapy after operation for carcinoma of the breast. This study consists of 117 female patients who were treated with a 4 mev. linear accelerator to a 15×5 cm. field including the scalene, supraclavicular and axillary lymph node areas. Thirty-three cases re-

ceived 6,300 rads in 12 increments over a 25 to 26 day period; 84 cases received 5,776 rads in 11 or 12 increments over a 25 to 26 day period. In the first group, 73 per cent exhibited neurologic symptoms, while in the second group only 15 per cent were found to have a neurologic deficit. These changes occurred within 5 to 30 months after the initial course of irradiation. The higher dose group tended to show initial symptoms somewhat earlier than the lower dose group. The presenting symptom in most cases was paresthesia or hypoesthesia of the fingers of the hand on the side of the treatment. Neurologic examination, however, revealed motor weakness as often as sensory changes. Usually a mixed motor and sensory deficit was found. In the milder cases, the symptoms tended to improve spontaneously after 6 to 9 months. Even in one of the most severe cases, there was electromyographic evidence of partial recovery after a period of 2 years.

The pathologic change was found to be a fibrous thickening of the neurilemma sheath, associated with demyelination and fibrous replacement of some of the nerve fibrils.

The authors emphasize the fact that similar symptoms have occasionally been seen even after quite modest doses in the order of 3,500 to 3,700 rads in 20 increments given over a 25 to 27 day period when utilizing orthovoltage therapy. Also, since many of their patients (34 per cent) had concomitant edema of the arm, it was suggested that pressure on peripheral nerves by edema may be an additional factor but definitely not the basic cause of the neuropathy.

The authors conclude that high dose, small field megavoltage irradiation following radical surgery leads to delayed damage of the brachial plexus, the incidence being related to the level of the dosage employed.—*Kenneth M. Nowicki, M.D.*

SANTÍN, GUILLERMO, and VARGAS S. JORGE. Roentgen study of cysticercosis of central nervous system. *Radiology*, March, 1966, 86, 520-528. (Address: Dr. Santín, Head, Department of Radiology, Hospital Francés de México, México, D. F.)

Cysticercosis is produced in man through the ingestion of ova of *Taenia solium*. The active oncosphere is liberated in the stomach; it then will penetrate the intestinal wall, entering the blood stream to be carried by the blood stream into the head, preferring the cerebrospinal fluid, pia-arachnoid, and brain. The incidence is reported as 3.36 per cent in 2,767 autopsy cases.

The roentgenologic manifestations are described and listed in terms of direct and indirect signs. They are: (a) Direct: (1) calcified scolex and cyst; (2) calcified portions of cyst; (3) soft tissue masses (larvae); (b) Indirect: (1) hydrocephalus; (2) spread sutures; (3) intrasellar erosion; (4) extrasellar erosion; (5) fibrinous septa in ventricles (ependymitis).



Intracranial calcification was present in 83 per cent of the cases and does not exclude viable larvae of active disease. Hydrocephalus was the most common indirect sign and is usually due to obstructing cyst formation, without displacement deformities as seen with neoplasia. These larvae cysts may be found anywhere in the cerebrospinal fluid pathway but are most common within the ventricles.

The clinical manifestations of cysticercosis are quite variable. The "plain" skull roentgenograms should suggest the diagnosis if the character of the intracranial calcifications is appreciated, but pneumoencephalography, ventriculography, and myelography are frequently indicated. The differential diagnosis should include: other parasites, tuberous sclerosis, neoplasia, toxoplasmosis, tuberculosis, and neurofibromatosis.

The authors have observed that the worst type of cases, with the poorest prognosis, are those with widespread intracranial calcifications.—*John Bond, M.D.*

#### SKELETAL SYSTEM

GOLDBLOOM, RICHARD B., STEIN, PAUL B., EISEN, A., MCSHEFFREY, J. B., BROWN, B. ST. JOHN, and WIGLESWORTH, F. W. Idiopathic periosteal hyperostosis with dysproteinemia: a new clinical entity. *New England J. Med.*, April 21, 1966, 274, 873-878. (From: Pediatric Medical Service and Departments of Radiology and Pathology, Montreal Children's Hospital, and Departments of Paediatrics, Radiology and Pathology, McGill University, Montreal, Quebec, Canada.)

The authors report the cases of 2 unrelated children, a 14 year old boy and a 10 year old girl, who suffered from a transient illness characterized by fever, bone pain and tenderness, inability to walk, radiographic evidence of subperiosteal new bone formation and dysproteinemia. In one patient the subperiosteal new bone formation was observed in the mandible, both humeri, both ulnae, the second and third metacarpals on each side, the femora, tibiae, fibulae, the first metatarsals, and the left fifth metatarsal. In the other patient there was involvement noted in the ulnae, femora and, questionably, the fibula and the tibiae. The serum protein disturbance was characterized by hypoalbuminemia and marked elevation of the alpha 2 and gamma globulins. A search of the literature did not reveal any previous reports of patients with this particular combination of findings.

The fever lasted 3 weeks in one patient and 2 months in the other. As the fever subsided, the roentgenographic evidence of periosteal hyperostosis gradually disappeared, and the electrophoretic pattern of the serum proteins returned to normal.—*Donald Widman, M.D.*

MORGAN, TREFOR, and STEPHEN, DAVID. Bone changes in urticaria pigmentosa: a report of a case. *Australasian Radiol.*, Feb., 1966, 10, 64-67. (From: Royal Prince Alfred Hospital, Sydney, Australia.)

A 24 year old man had long-standing skin lesions typical of urticaria pigmentosa. In addition he had hepatosplenomegaly, generalized lymphadenopathy, and generalized increased density of bone. Bone marrow biopsy showed myelofibrosis and osteosclerosis, but no increase in mast cells. Liver and lymph node biopsies were normal and also not diagnostic for systemic mastocytosis. Splenectomy, necessitated because of pressure symptoms, revealed excessive mast cells.

The difficulty in diagnosing systemic mastocytosis histologically has been experienced by others and is apparently due to the propensity of the mast cell to disintegrate.

Bone involvement is demonstrable in 20-40 per cent of the cases extensively investigated, and if followed longer, the incidence may be higher. The bone changes which are confined almost to cancellous bone represent the result of tissue mast cell hyperplasia with fibrosis and later sclerosis of bone marrow. Generalized increase in density due to thickened trabeculae is the common appearance, at times accompanied by cortical thickening and periosteal reaction. Gastrointestinal involvement does occur but is less common than the bone changes. The bone abnormalities are usually asymptomatic.—*Mark D. Reiss, M.D.*

MACHTEY, ISRAEL, RODNAN, GERALD P., and BENEDEK, THOMAS G. Paget's disease of the hip joint. *Am. J. M. Sc.*, May, 1966, 251, 524-531. (From: Department of Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pa.)

The problem of hip joint disease due to osteitis deformans has received relatively little attention in the American literature, although this aspect of Paget's disease has been treated rather extensively in European publications. The authors state that during the past few years they have become increasingly impressed with the importance of hip involvement in patients with Paget's disease and have carried out studies in order to secure better understanding of the clinical and roentgenographic manifestations of this disorder and to obtain some estimate of its frequency.

The clinical records and roentgenograms of 98 patients with Paget's disease of bone were reviewed with particular attention to the occurrence of hip joint involvement. Ten patients were found to have definite evidence of coxopathy, the chief features of which were marked concentric narrowing of the "joint space," and bony lipping of the acetabulum which was variably deepened and deformed. In 11

other cases, in which the roentgenograms were not sufficient for adequate evaluation, there were changes which were considered suggestive of this hip disorder. The majority of the patients with Paget's coxopathy complained of pain in the hip and a limp and were found to have limitation in motion of the joint.

These changes in the hip joint appear to be the result of weakening and deformation of the bony constituents of the joint.—*C. Peter Truog, M.D.*

LANGLEY, G. R., SABEAN, H. B., and SORGER, K. Sclerotic lesions of bone in myeloma. *Canad. M. A. J.*, April 30, 1966, 94, 940-946. (From: Departments of Medicine, Radiology and Pathology, Dalhousie University and the Victoria General Hospital, Halifax, Nova Scotia, Canada.)

The authors record in detail the findings in 5 patients in whom a sclerotic reaction of bone was observed in association with myeloma.

The absence of skeletal sclerosis in myeloma is familiar. It is reflected in the sharply demarcated lytic lesions without osteoblastic reaction and the usually normal serum alkaline phosphatase. Osteoblastic proliferation can occur however, since adequate callus may form at the sites of pathologic fractures, and dense sclerosis has been observed after irradiation or other therapy. The occurrence of sclerosis in proved myeloma, in the absence of fracture or therapy, has been recorded in 18 patients. More often, however, single or multiple focal areas of sclerosis, sometimes in association with osteolysis, osteoporosis, or spicule formation, have been seen.

The association of myeloma with myelosclerosis, myelofibrosis and polycythemia vera has led to the suggestion that myeloma be included among the myeloproliferative disorders, implying that a totipotent stem cell may be capable, under appropriate stimulation, of differentiating into one or more of several cell types.

The overlapping histologic features and the anomalous proteins found in multiple myeloma, lymphosarcoma and lymphocytic leukemia suggest that myeloma be classified in close relationship to the lymphoproliferative states.

The concept of myeloma as a myeloproliferative disorder would indicate that the sclerosis of bone in these patients was the result of a simultaneous proliferation of bone-forming elements leading to increased bone deposition. Of 4 patients in this series in whom bone was available for examination, there was no histologic evidence of active bone formation. The serum alkaline phosphatase was elevated in 1 patient and normal in 2 others. Therefore, the possibility of some other mechanism, such as interference with the normal resorptive processes, possibly as a result of neoplastic infiltration of the marrow

spaces, cannot be excluded as a cause of the osseous sclerosis.

Bone sclerosis can be observed in a vast number of diseases in addition to myeloma. It is important to determine whether there was a coincidental association of sclerosis in these patients with myeloma or if there was some relationship between the two processes. It is not possible to exclude the former completely or to provide unequivocal evidence of the latter.

The earlier recognition of myeloma, brought about particularly through the increasing use of serum electrophoresis, has enabled the diagnosis to be established, frequently before radiologic changes are evident.—*Stephen N. Tager, M.D.*

#### BLOOD AND LYMPH SYSTEM

BRENNER, H., and ZAUNBAUER, W. Über Fehlermöglichkeiten bei der zerebralen Angiographie. (Possibilities for error in cerebral angiography.) *Radiologia Austriaca*, 1966, 16, 45-52. (Address: Dr. Heinrich Brenner, I. Chirurgische Universitätsklinik, Allgemeines Krankenhaus [I. Hof.], Wien IX, Austria.)

Faulty technique may be the source of error in cerebral angiography. The films should be large enough (10×12 cm.) to include the injection site, so that it can be immediately determined if an intramural or paravascular injection has been made. The authors avoid the use of tubes and automatic injector systems since they feel that they can more easily recognize alterations in resistance within the vessels and make the necessary adjustments manually. Simultaneous, biplanar, serial exposures are essential, particularly in the diagnosis of aneurysms.

Errors in interpretation may be made if examination is undertaken soon after bleeding from an aneurysm. Spasticity of neighboring arteries may interfere with adequate opacification of the aneurysm and lead to erroneous diagnoses. In such cases, re-examination after a short interval may produce a satisfactory demonstration of the aneurysm. The authors also describe one angiogram which was erroneously interpreted as a vascular anomaly but surgery disclosed a meningioma with bizarre and markedly dilated vessels.

Stenosis of the anterior cerebral artery is rare unless it is associated with an anomaly of the circle of Willis. Changes in the carotid artery may be responsible for a faulty diagnosis of stenosis of the anterior cerebral artery. In increased intracranial pressure (cerebral tamponade), occlusion of this vessel may be misdiagnosed, since a massive hematoma may produce spasm of this vessel.

Angiography may be misleading in the diagnosis of tumors. One instance is cited in which characteristic staining suggested a tumor but on surgical intervention, an area of cerebral softening was found. In



another case, the angiographic studies were considered normal but, when the patient's condition deteriorated, an operation was performed and a glioma was found. A correct diagnosis may be difficult when two separate conditions are present simultaneously, since the changes due to one condition may hide or alter the expected findings due to the other pathologic entity. Biplanar studies are recommended because it is possible that a lesion may be missed if studied in only one view, lateral or frontal. Serial studies are highly recommended so that a given lesion may be studied in every phase. Repeat or follow-up studies are often fruitful in eliciting pathology despite negative findings on the initial survey, especially in post-traumatic cases, since the local vascular picture may change from day to day.—*J. Zausner, M.D.*

**VOGELSANG, H.** Möglichkeiten von Fehlbeurteilungen beim Hirnabszess durch die Karotisangiographie. (Possibilities for incorrect diagnosis of brain abscess by means of carotid angiography.) *Radiologia Austriaca*, 1966, 16, 63-68. (Address: Klinikstrasse 37, Giessen/Lahn, Germany.)

Various reasons for the failure of carotid angiography to establish the diagnosis of brain abscess are listed:

(1) Angiography is normal. One patient, treated with antibiotics for a brain abscess, had normal findings on angiographic studies but when the condition deteriorated, surgical intervention disclosed an abscess in the temporal region. Another patient had mildly tortuous vessels and the condition was diagnosed as an anomalous vascular process but subsequent surgery showed bilateral parietal abscesses.

(2) Unusual location of an abscess, such as in the basal ganglia, cerebello-pontine angle, fronto-medial portion of the brain, adjacent to the ventricular system, a small abscess near a thickened suture, or within an atrophic area, may be associated with a normal angiogram and failure to make the correct diagnosis. One case is cited in which a small abscess had perforated into the ventricular system, so that a "space-occupying" lesion was not demonstrable by angiography.

(3) Atypical staining (characteristic of a cystic tumor) led to an error in diagnosis. A diagnostic tap enabled establishment of the true nature of the lesion.

(4) Technical difficulties (see abstract of the article by H. Brenner and W. Zaunbauer).

(5) Anomalies and thickened sutures may also be responsible for an incorrect diagnosis.—*J. Zausner, M.D.*

**MAURER, H.-J., FRIELINGS DORF, D., and VLEUGELS, E.** Möglichkeiten der Fehldeutung im Splenoportogramm. (Possibilities for in-

correct diagnosis in splenoportography.) *Radiologia Austriaca*, 1966, 16, 69-75. (Address: Prof. Dr. H.-J. Maurer, Röntgenabteilung der Chirurgischen Universitätsklinik und Poliklinik, 53 Bonn-Venusberg, Germany.)

Splenoportography is performed when the patient's basic condition is compensated and operable. The examination is done with the aid of the Gidlund injector. Electrocardiographic tracings are taken simultaneously. The patient is under mild anaesthesia. The chief indication for this procedure is portal hypertension as in hepatic cirrhosis. The authors rarely use this method for other liver diseases, such as parasitic infestations, abscesses or metastases.

Errors in evaluation of the intrahepatic vascular tree are rare. The main sources of errors are divided into three groups:

(1) Improper evaluation of positive findings. This is due to failure to recognize irregularities of the terminal portions of the intrahepatic vascular tree. One case is cited in which gastric varices were overlooked, so that the correct diagnosis could not be made.

(2) Failure to recognize the full extent of anastomoses between the splenoportal and caval systems. Such failures may account for some poor results and some of the complications following surgery.

(3) Inaccurate interpretation of the confluence sign and mural thromboses. A case of extensive thrombosis of the splenic vein is reported. The diagnosis was almost missed because the injection needle had been improperly placed in the superior pole of the spleen. The spleen had been displaced by marked enlargement of the left lobe of the liver. Surgical intervention was indicated and the thrombi were successfully removed.—*J. Zausner, M.D.*

**RÜTTIMANN, A.** Fehlermöglichkeiten bei der Lymphographie. (Errors in lymphography.) *Radiologia Austriaca*, 1966, 16, 77-86. (Address: Röntgendiagnostisches Zentralinstitut der Universität, Kantonsspital, Zürich, Switzerland.)

Errors in lymphographic studies are either due to poor technique or faulty interpretation.

To avoid technical errors, the examination should be done with fluoroscopic control and the injection should be made under constant pressure, preferably below 0.4 atm.

Lymphography affords a study of the morphology and function of the lymphatic system.

For proper evaluation of the findings, it is essential that the examiner know the history of the patient, especially with reference to edema, infection, trauma or abdominal surgery. The radiologist should also be familiar with the clinical picture, e.g., whether an inguinal hernia is present or not (palpation by the

radiologist is highly recommended). An inguinal hernia may displace the lymph nodes and thus simulate a tumor.

The radiologist need not attempt to make a histologic diagnosis, since it is very difficult sometimes to distinguish between degenerative changes and early metastatic involvement. Likewise the presence of one lacuna does not necessarily mean metastasis.

Mild degrees of fibrosis rarely lead to obstructive phenomena but widespread fibrosis produces dilatation of lymphatic channels, stasis of the contrast material, and subsequent collaterals within the lymphatic system.

If the initial study is negative or indicates a benign process, re-examination should be done after a suitable interval if there is the slightest suspicion of a neoplasm (the author suggests 2-3 months).—*J. Zausner, M.D.*

**BRYANT, LESTER R., and SPENCER, FRANK C.**  
Occlusive disease of subclavian artery.  
*J.A.M.A.*, April 11, 1966, 196, 123-128. (Address: Dr. Bryant, University of Kentucky Medical Center, Lexington, Ky.)

Fourteen patients whose primary symptoms were due to subclavian artery occlusive disease were evaluated clinically and by arteriography. Seven complained only of ischemic symptoms of an upper extremity, 3 only of vertebral-basilar insufficiency, and 4 had both symptom patterns. Contrast visualization of the aortic arch was obtained in 12 of the 14 patients. In 1 of the 2 patients in whom arteriography was omitted, the partially obstructed vessel was an aberrant retroesophageal right subclavian artery, the lack of this knowledge making the surgical procedure considerably more difficult.

Characteristically, occlusive disease of the right subclavian artery began at its origin from the innominate artery and involved a segment 2 to 4 cm. in length. Occlusive disease of the left subclavian artery was also present primarily at the origin of this vessel from the aorta, but 2 of 6 patients showed disease beginning distally with involvement of the origin of the left vertebral artery.

Subclavian steal was noted in 7 of the 12 patients. A difference in blood pressure of 20 mm. Hg or more in the two arms was noted in all patients in this series. Those with primary symptoms of upper extremity ischemia showed differences of 50 mm. Hg or more. Those with cerebral ischemia were under this figure, suggesting that in these patients the vertebral artery delivers to the arm a significant amount of blood intended for the cerebral circulation.

The authors emphasize that patients with similar angiographic findings may have different symptoms. They also point out that arteriography is necessary for the selection of a proper surgical approach for relief of the occlusion: a high position of the innominate bifurcation may allow a supraclavicular ap-

proach; otherwise, a midline sternal incision or a posterolateral thoracotomy is required.—*Stephen A. Kieffer, M.D.*

**VIGNE, J., DEBONNIÈRE, C., GARRETA, L., and KLEIN, P.** Le syndrome de Pegot-Cruveilhier-Baumgarten. (The Pegot-Cruveilhier-Baumgarten syndrome.) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1965, 46, 763-764. (From: Services de Première Médecine and d'Electroradiologie de l'H.M.I., Val-de-Grâce Paris, France.)

This syndrome consists of the combined association of the abnormal persistence of the umbilical vein and a cirrhosis of any etiology, together with portal hypertension.

The disease bearing the same name is characterized by a primary anomaly of the portal system with permeability of the umbilical vein and a peri-umbilical subcutaneous tumor. In the original description the liver was normal.

Clinically, the syndrome may be suggested if there are voluminous peri-umbilical varices. If these varices are not noticed clinically, they will be revealed by splenoportography.

The authors report a case of this syndrome in a 54 year old male with a history of ethylism, hepatosplenomegaly, and para-umbilical varices without ascites or esophageal varices. The case was investigated by means of splenoportography and laparoscopy. A hepatic biopsy revealed an atrophic cirrhosis.—*H. P. Lévesque, M. D.*

**FONKALSRUD, ERIC W., LINDE, LEONARD M., and LONGMIRE, WILLIAM P., JR.** Portal hypertension from idiopathic superior vena caval obstruction. *J.A.M.A.*, April 11, 1966, 196, 129-132. (Address: Dr. Fonkalsrud, Department of Surgery, Center for the Health Sciences, University of California, Los Angeles, Calif.)

The combination of extensive idiopathic mediastinal and retroperitoneal fibrosis in a child resulted in obstruction of venous return to the heart with both systemic and portal venous hypertension. Jugular venography demonstrated marked dilatation of the brachiocephalic, jugular, and subclavian veins with large dilated collateral channels extending to the chest wall and eventually to the azygos system. Femoral venography showed complete obstruction of the common iliac veins and the inferior vena cava. The portal hypertension was thought to be due to obstruction of the hepatic veins at their junction with the inferior vena cava.

Portal hypertension had resulted in bleeding esophageal varices and marked ascites which was refractory to medical management. Portal decom-



pression was undertaken as a lifesaving measure, but because of the obstructed inferior vena cava, the usual surgical procedures, *e.g.*, portacaval or spleno-renal shunt, would have been of no value. A novel procedure was therefore devised, involving insertion of a teflon tube graft between the splenic artery and the left lower lobe pulmonary artery. There was immediate significant fall (39 to 32 cm. of water) in pressure in the splenic vein, and the patient is much improved symptomatically on follow-up 9 months after the procedure.—*Stephen A. Kieffer, M.D.*

**DOTTER, CHARLES T., FRISCHE, LOUIS H., JUDKINS, MELVIN P., and MEULLER, ROBERT.** The "nonsurgical" treatment of iliofemoral arteriosclerotic obstruction. *Radiology*, May, 1966, 86, 871-875. (Address: Department of Radiology, University of Oregon Medical School, 3181, S.W. Sam Jackson Park Road, Portland, Ore.)

The authors present a progress report on the technique of transluminal recanalization of arteriosclerotic obstructions with particular emphasis on the problems encountered, the current criteria for case selection, and areas of future promise.

The technique of transluminal recanalization is basically that of a probe directed through a narrowed or obstructed lumen, followed by radial displacement and molding of the atheromata to re-create or widen a vessel lumen. Such an approach tends to eliminate the rough lumen and abrupt changes in caliber which predispose to thrombosis after endarterectomy or bypass grafting. The procedure is usually performed under local anesthesia and, after appropriate percutaneous needle entry, a coil spring guide is introduced. The guide is maneuvered across the obstructed segment with the assistance of image intensification fluoroscopy. Thereafter, tapered radiopaque teflon dilating catheters are passed coaxially over the coiled spring guide, enlarging the path of the guide wire. A dilute contrast medium is used intermittently during the dilatation and following its completion for both fluoroscopic and conventional arteriographic assessment of progress. All instruments are withdrawn at the conclusion of the procedure and hemostasis is achieved with manual compression over the entry site. After a successful recanalization, patients are given daily intravenous infusions of dextran for 3 days and encouraged to walk immediately.

The present series includes 113 procedures on 74 patients but, since several of the patients had bilateral lesions, 82 different limbs or individual lesions are represented. The majority of lesions were located to the femoro-popliteal system but 7 were in the posterior tibial arteries and 6 involved the iliac arteries. Numerous patients had frank gangrene for which amputation appeared the only recourse and the contemplated amputation was averted in at least 8 in-

stances. The results are encouraging, both as to the immediate and long-term effect on the limb circulation as the symptoms and signs were successfully improved in 32 patients. In 37 patients, no appreciable change was noted and in only 3 did the condition appear worsened.

The most difficult problem encountered during transluminal recanalization of a totally occluded vessel is that of a coiled spring guide either entering a peri-atheromatous cleavage plane or perforating the vessel wall. Although neither endangers the patients, such may preclude the lumen-to-lumen traverse which is essential to success. Numerous techniques have been tried to overcome this difficulty, including gas as a dissecting agent and a special long blunt-ended hypodermic needle, but none has been entirely satisfactory. The greatest success with the technique will be obtained in patients with arterial narrowing, rather than complete occlusion, as the residual lumen provides a readily followed route for the spring guide. In such a situation, transluminal therapy should be successful 90 per cent of the time with a reasonable prospect of lasting benefit.

Ideally, transluminal dilatation should be employed in an attempt to prevent the development of severe arterial obstruction and attendant ischemia of a limb. From a practical standpoint, the procedure is strongly indicated in impending or actual gangrene, regardless of whether the responsible occlusion is incomplete obstruction or total. It may be used after a surgical failure, such as endarterectomy or bypass grafting, and can be repeated in a previously dilated vessel presenting additional areas of narrowing.

Much remains to be done in exploring the future possibilities of this technique but progress has been made in application to sites other than the leg arteries. Several successful instances of iliac artery recanalization are presented by the authors and a recently fabricated reinforced balloon-type dilator is illustrated. With newer instrumentation, the renal and brachiocephalic arteries represent a promising area for future application and transluminal therapy has recently been used in the transaortic treatment of coronary atherosclerosis, an area which probably offers the greatest potential of all.—*Edward B. Best, M.D.*

**JACKSON, REGINALD J. A.** Complications of lymphography. *Brit. M. J.*, May 14, 1966, 1, 1203-1205. (From: Department of Obstetrics and Gynaecology, Queen Elizabeth Hospital, Birmingham, England.)

The minor complications of lymphography are well known and have been extensively described in the literature. These are transient fever, cough, malaise, nausea, tachycardia, and local infection on the dorsum of the foot. Oil embolization is commonly seen on chest roentgenograms, and may be asymptomatic or cause fever and cough. Several severe re-

actions have been seen, and 4 deaths attributed to oil embolization have been reported. The development of untoward effects depends on the amount of contrast medium which directly reaches the lungs, and on the presence of pre-existing pulmonary disease. The dosage must be lowered in patients with demonstrated lymphatic obstruction, as an increased incidence of embolization is seen in these cases, presumably due to lymphatico-venous anastomoses.

The complications of 110 consecutive successful lymphangiographies done on females with genital carcinoma were evaluated. Minor complications were seen in 30 per cent. Three patients developed deep venous thromboses of the legs and pulmonary thromboembolism. All 3 had extensive malignant disease of the pelvis, and since there is a well known relationship between pulmonary thromboembolism and extensive malignancy, the association with lymphography may be coincidental. However, the venous stasis occurring over the several hours during the examination probably contributed to the complication. Soft foam mattresses and periodic passive exercise have been utilized since. Six patients had hemoptysis. This probably resulted from breakdown of oil and release of fatty acids in the lungs.—*Mark D. Reiss, M.D.*

**DOLAN, PATRICK A.** Lymphography: complications encountered in 522 examinations. *Radiology*, May 1966, 86, 876-880. (Address: Department of Radiology, Methodist Hospital of Indiana, Indianapolis, Ind.)

The author reports a high incidence and wide variety of complications, mostly minor and of short duration, encountered during or after intralymphatic injection of ethiodol for lymphography in 522 examinations. A total of 119, or 26 per cent of these patients had complications apparently related to the ethiodol injected.

The most common complication, both singly and occurring concurrently with other reactions, was a low-grade febrile response beginning about 4 hours after the injection and lasting 4-6 hours. This is thought to be a pyrogenic systemic response to the contrast medium. Over half of these 97 patients had roentgenographically demonstrable oil micro-emboli in their lungs which, in the great majority, were no longer visible after a maximum of 3 days and caused the patients no distress. However, 1 of this group developed frank bilateral bronchopneumonia 24 hours after the injection and another (a child) developed pulmonary densities with fever, wheezing and coughing which persisted for about 1 week.

Nausea and/or vomiting occurred with 23 patients, usually soon after the injection and transitory in nature. Seventeen patients experienced pain in the injected limb, groin, or back during or after injection. The majority were relieved upon decreasing the injection pressure but a few with obviously abnormal

lymph nodes, particularly those with lymphoma, experienced constant dull groin or back pain for up to 24 hours after the injection. Seven patients developed respiratory difficulties, 5 of whom had transitory symptoms consistent with mild bronchospasm. The other 2 mentioned above developed pneumonia or a pneumonia-like condition. Life-threatening reactions occurred with 2 patients—1 a hypertensive episode and the other an anaphylactic hypotensive reaction. Seven patients had localized reactions attributable to the iodine in ethiodol—2 developed iodine sialitis and 5 had a rash in the injected limb which were mainly of short duration, although 1 rash persisted for 4 weeks.

There were a number of problems involving factors of technique, including 13 cases of unsuccessful study due to failure to find a cannulable lymph vessel (usually due to pedal edema) or inability to maintain cannulation. In 1 case a vein was unintentionally cannulated. Local infections occurred in 5 patients, including a case of lymphangitis and 4 infected cut down sites.

The author did not find any permanent lymphatic, renal or pulmonary changes on repeat lymphography at a later date or at autopsy in some of these patients. He states that, although lymphography is time-consuming and associated with a high percentage of complications, most of these are minor and of short duration. In selected patients it is a valuable diagnostic and prognostic method and most of these examinations can be performed on an out-patient basis.—*Victor B. Brasseur, M.D.*

#### GENERAL

**SALOMONI, ENZO.** (Rome, Italy.) Méthode de Turano pour l'étude des fonctions motrices des viscères musculaires creux (exploration simultanée roentgencinématographique et des pressions). (Turano's method applied to the study of motor-functions of hollow muscular viscera [simultaneous roentgen cinematographic and pressure recording].) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1965, 46, 707-719.

In 1956 Turano devised, in the laboratory of the Radiological Institute of the Rome University, a method for the study of esophageal mobility which was soon adapted to the exploration of motor-functions of all hollow muscular viscera.

This method consists of the simultaneous recording of intravisceral pressures and radiocinematography. It allows the observation of the morphologic and dynamic aspects of each phenomenon occurring in the hollow muscular viscus under investigation.

After describing the technique and its use, the author illustrates the salient results obtained during investigations of the digestive tract, the biliary tree, the urinary apparatus and the female genital tract.



He presents the advantages and the potentiality for analytic studies offered by the synchronized combination of radiocinematography and pressure recording. Finally, he discusses the limitations inherent on the use of this method.—*H. P. Lévesque, M.D.*

KOSLOWSKI, L., and WELLER, S. The application of x-ray image intensification to orthopaedic surgery. *German Med. Monthly*, Feb., 1966, 11, 61-62. (Address: Chirurgische Universitätsklinik, Hugstetter Str. 55, Freiburg/Br., Germany.)

The authors advocate the use of mobile image intensification units in orthopedic surgery. These should rotate along a semicircle about a horizontal and vertical axis, making them highly versatile for fluoroscopy and radiography. However, they admit that care must be used not to turn on the image intensifier more frequently or for longer periods than necessary.

They have used this apparatus in repositioning fractures of the arm, hand, femur, lower leg and calcanei. They have also employed it in percutaneous pinning of bones of the arm and leg and femoral neck fractures. With this technique the fracture is not exposed and infection is reduced to a minimum.—*Arthur E. Childe, M.D.*

WISE, ROBERT E., and GANSON, JORGE. Subtraction technic: video and color methods. *Radiology*, May 1966, 86, 814-821. (Address: Department of Diagnostic Radiology, Lahey Clinic Foundation, 605 Commonwealth Avenue, Boston, Mass.)

The authors describe refinements of the subtraction technique with television cameras and with color methods and discuss their advantages and disadvantages.

The video method uses 2 television cameras of the Vidicon type, with the polarity of one camera reversed to create a diapositive image—the two images are then superimposed on a single monitor, to produce a subtracted image.

In the authors' experience, the video method presents several difficulties: (1) it is time-consuming, requiring numerous electronic adjustments, and (2) the size of the field in which perfect superimposition of images can be obtained is only  $\frac{1}{4}$ – $\frac{1}{2}$  of a 10×12 inch roentgenogram. However, this technique enhances contrast, often showing vessels clearly which are barely perceptible to the unaided eye. This has been particularly useful to the authors in abdominal aortography and in cerebral angiography where confusing bone shadows may be eliminated. The method has been of little value in enhancing tumor stains. It frequently helps to enhance contrast in cardiac angiography, especially in the left cardiac chambers.

Color subtraction depends on production of white

light by the addition of primary colors or the production of a third color from 2 primary colors. Roentgenograms, viewed on 2 viewboxes, one producing a red light source, the other a blue light source, are seen as a composite image through a beam-splitting mirror. Arteries are portrayed in red, and veins in blue, and the background structures in neutral tones.

The authors state that the video system is superior in its ability to enhance contrast, and is easily photographed with Polaroid film for rapid evaluation of examinations. It is much more expensive than the color technique however, and requires lengthy pre-examination adjustments of the video apparatus. The color method, on the other hand, is valuable as a teaching medium, and superior in superimposition, reliability, and simplicity.—*James A. Cunyus, M.D.*

TEN CATE, HARRY W., JONGMANS, H., and ZIESES DES PLANTES, B. G. Subtraction technique in renal angiography. *J. Urol.*, March, 1966, 95, 421-428. (From: Section of Urology, Department of Surgery and the Department of Radiology, University of Amsterdam Medical School, Amsterdam, Netherlands.)

The subtraction method applied to radiographic technology is a procedure of value which under certain conditions eliminates unessential shadows. It can be readily applied to renal angiograms with resulting improvement in visualization of the important structures to be studied.

To obtain good results with the subtraction technique, two films are necessary which are the same excepting for the presence of contrast medium in one of them. Prior to aortography, a survey roentgenogram is made of the renal area under standard technical conditions with careful attention of the phase of respiration. From this film, a diapositive print is made.

The renal angiogram is made by the customary method with great care to maintain the patient's position and stage of respiration similar to that of the original survey roentgenogram. The actual subtraction is done by placing the arteriogram on a glass plate beneath the diapositive. Both films are carefully superimposed in the darkroom and a third print is then made by exposure to a measured amount of light. When this latter film is processed, it represents a final subtraction print. These prints are usually made with high contrast film or paper.

In this article, the authors show the value of the subtraction technique in renal angiography in patients who have renal artery lesions, renal cysts, and renal tumors. The subtraction method may be particularly helpful in those patients who are unusually heavy, in those who have superimposed bowel gas, or in those instances where an opinion on the fine arterial branches is needed. In the differential diagnosis of renal cyst and tumor, subtraction may clarify the

picture to such a degree that surgical exploration may be unnecessary.

The authors include 10 figures which illustrate the technique and its application.—*George W. Chamberlin, M.D.*

### RADIATION THERAPY

KAUFMAN, B., PEARSON, O. H., SHEALY, C. N., CHERNAK, E. S., SAMAN, N., and STORAASLI, J. P. Transnasal-transsphenoidal yttrium-90 pituitary implantation in the therapy of acromegaly. *Radiology*, May, 1966, 86, 915-920. (Address: Western Reserve University School of Medicine, Cleveland, Ohio.)

Yttrium 90 was introduced into the sella turcica via a transnasal, transsphenoidal approach in the treatment of 9 patients with active acromegaly. There was no evidence of suprasellar extension or optic nerve compression in any of these patients. Five had received previous external irradiation without lasting significant improvement. Pre-implantation roentgenograms and laminagrams were obtained to determine the volume and geometry of the enlarged gland. In markedly asymmetric enlargements, arteriography was employed to evaluate the lateral margins. Pneumoencephalography probably should be utilized in all patients to visualize the diaphragma sellae.

Under sterile conditions, the  $Y^{90}$  rods were placed in nylon jackets and implanted in the enlarged pituitary according to the screw implantation technique of Forrest. The rods were all 2 mm. in diameter and varied in length, with combination of 2 and 3 mm. lengths being employed.

At surgery, the patient's head is placed in a head-holder and the nose is packed with gauze soaked in a 50-50 mixture of 4 per cent cocaine and zephiran. Five minutes later, the cannulae are advanced to the floor of the sphenoid sinus with fluoroscopic monitoring. After a suitable angle is obtained, the floor or the sphenoid sinus is drilled and the cannulae are advanced to the floor of the sella. Roentgenograms in the lateral and posteroanterior projections are taken. When proper alignment is obtained, the floor is

drilled and tapped and the active sources and screw are inserted. All patients showed improvement as evidenced by objective tests such as urine calcium excretion, change in size of the hands and feet, serum phosphorus levels, serum insulin levels during glucose tolerance tests, and insulin and glucose tolerance. Serum growth hormone levels showed a decrease and subjective improvement was present in all patients.

There were few complications in this group of acromegalic patients. Cerebrospinal fluid rhinorrhea developed in 2 patients. Diplopia occurred in 1 patient. Damage to cranial nerves is a major reported complication and the late development of a malignant neoplasm, especially a sarcoma of the heavily irradiated tissue, remains a possibility. Meningitis may result with potentially fatal consequences.

It is concluded that interstitial irradiation with  $Y^{90}$  cannot regularly achieve ideal treatment of acromegaly. However, the response to therapy in this group of patients appears to be satisfactory, and this technique may have a place in the therapy of acromegaly in carefully selected patients.—*A. W. Sommer, M.D.*

SANDLER, HENRY C. Errors of oral cytodiagnosis: report of follow-up of 1,801 patients. *J. Am. Dent. A.*, April, 1966, 72, 851-854. (From: Dental Service, Veterans Administration Hospital, 800 Poly Place, Brooklyn, N. Y.)

The author assesses cytodiagnostic accuracy. Oral abnormalities were found in 2,758 patients.

There was a high degree of positive correlation between the biopsied specimens and the cytologic diagnosis.

The exceptions were as follows: (1) On cytologic examination there were 8 "false" negatives and 7 "false" positives; (2) 22 cases demonstrated positive cytodiagnosis with an initial negative biopsy. This signified a 2.5 per cent error with respect to cytodiagnosis and a 3.7 per cent biopsy error.

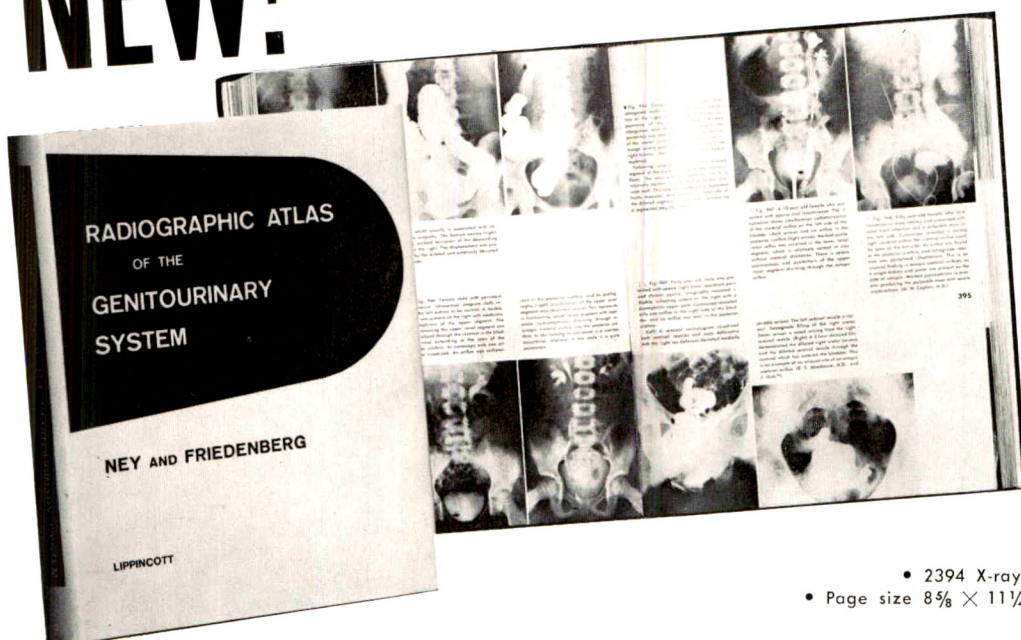
It is concluded that oral cytodiagnosis can be effective in the early diagnosis of oral cancers.—*Henry Haven, M.D.*





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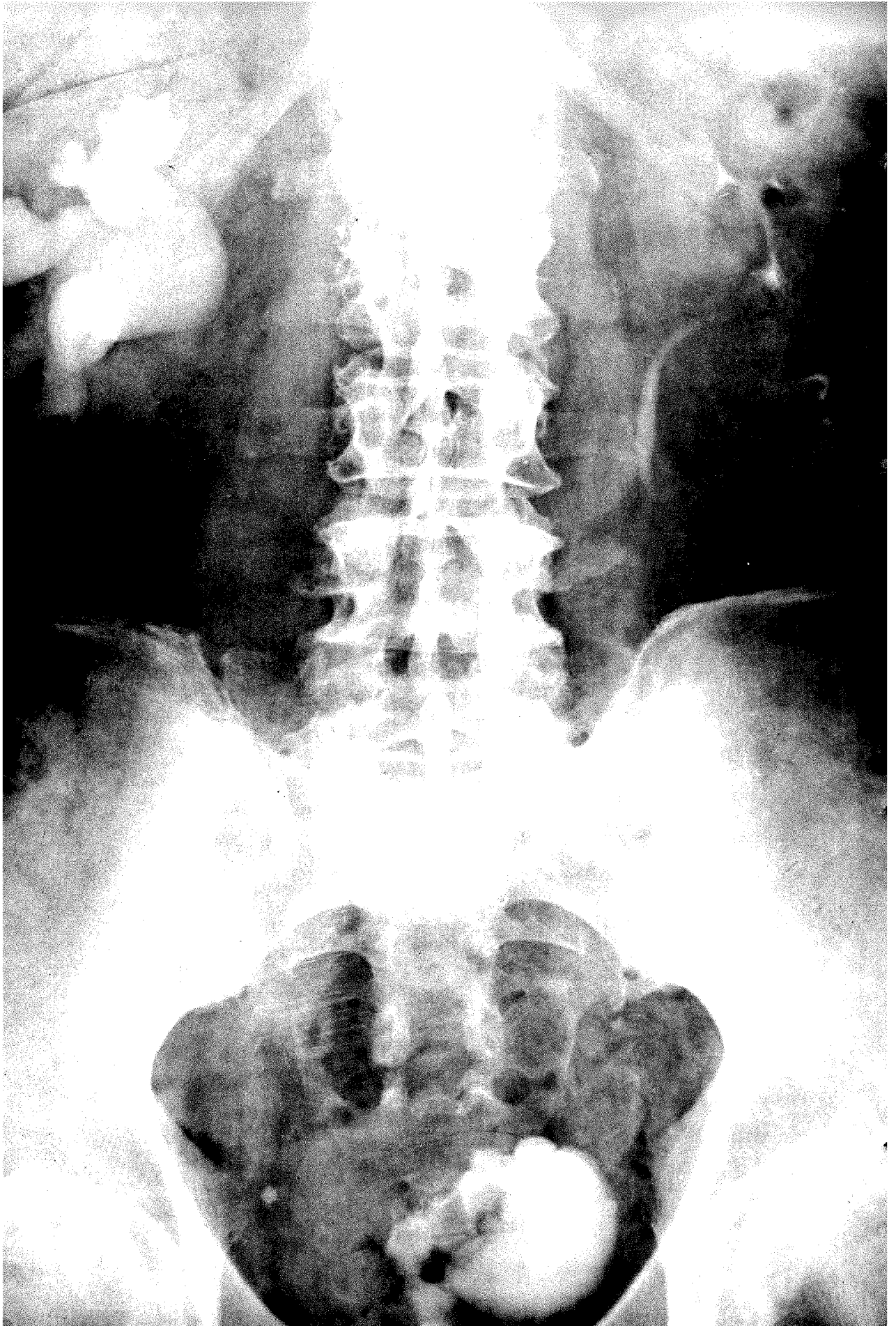
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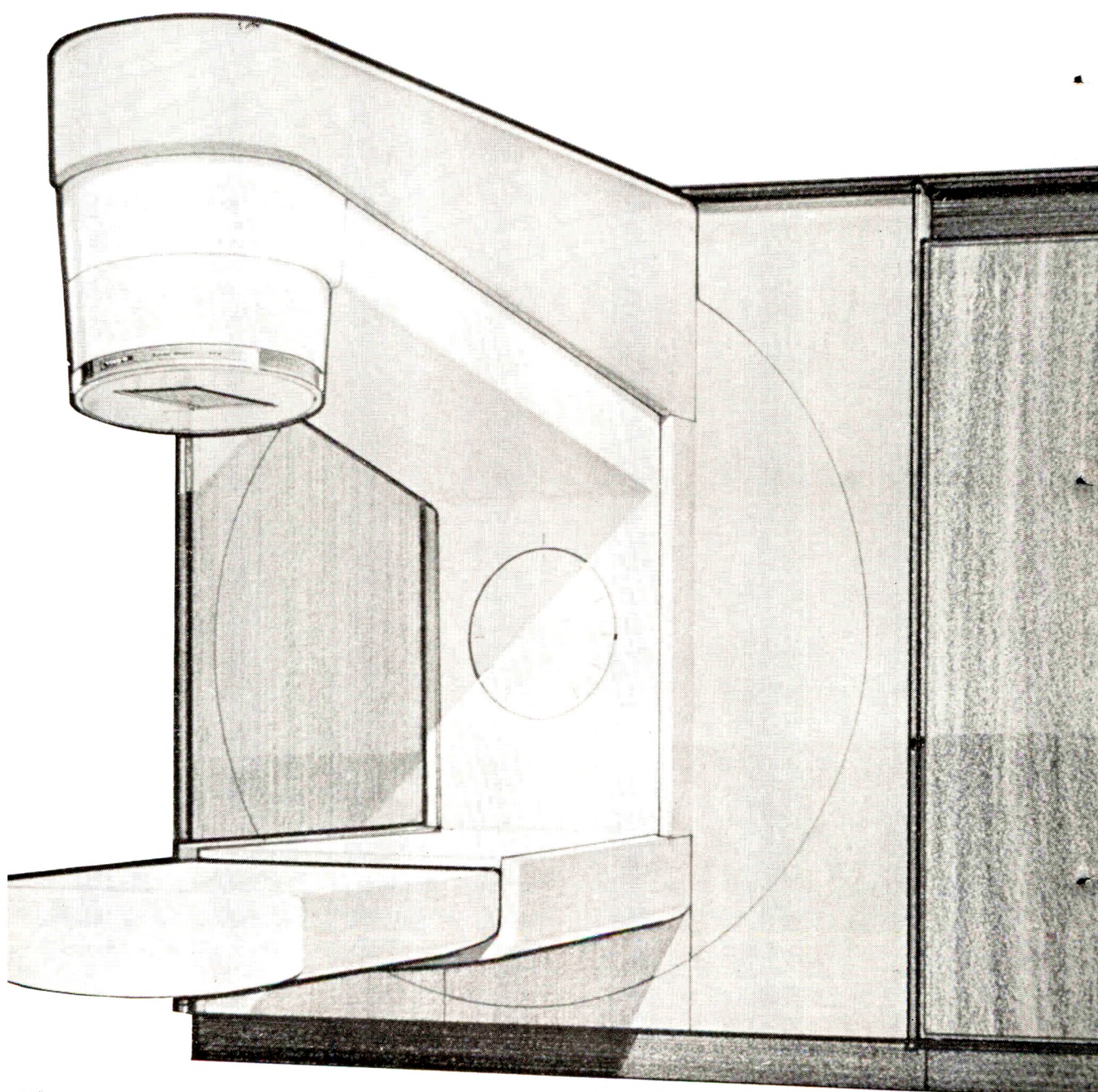
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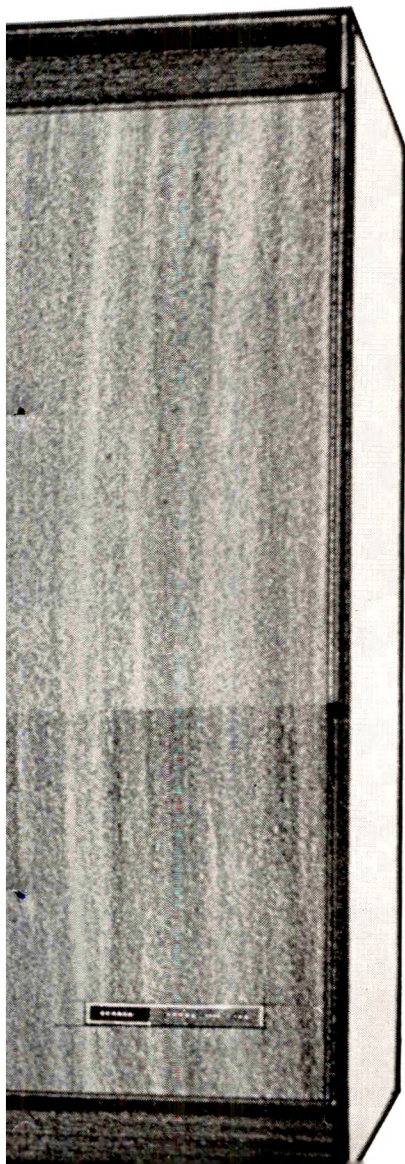
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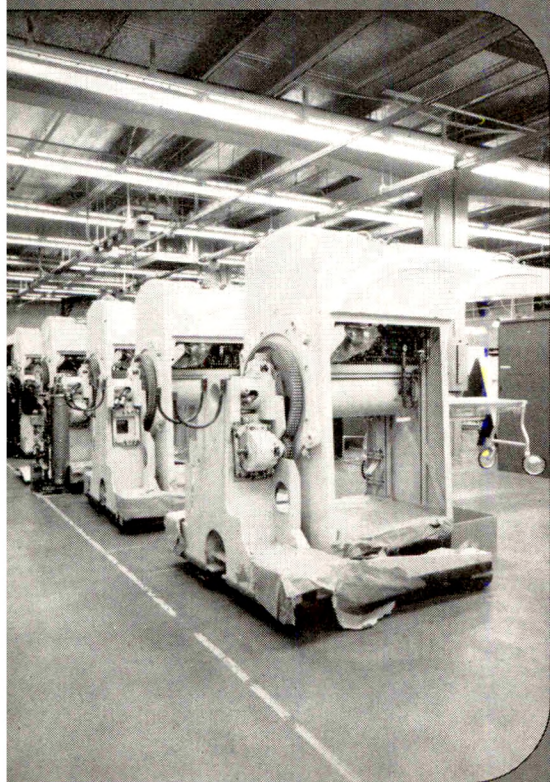
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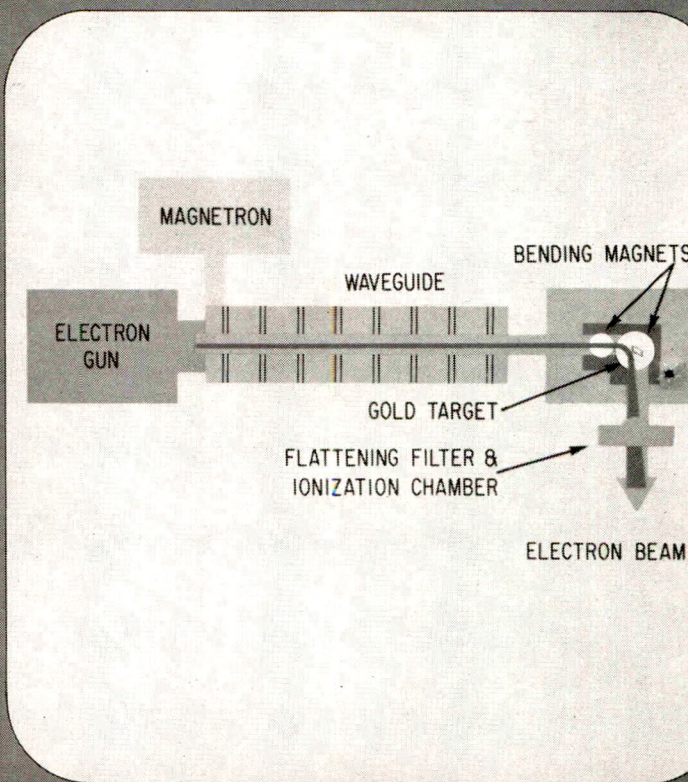


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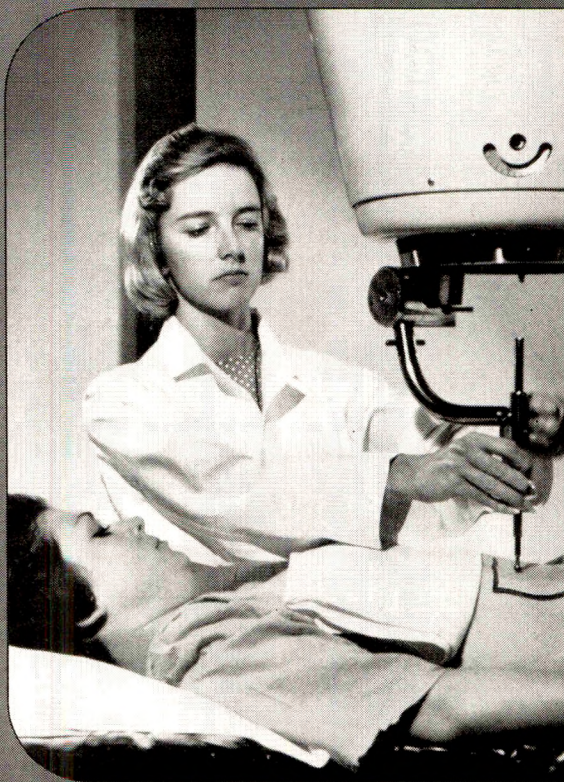
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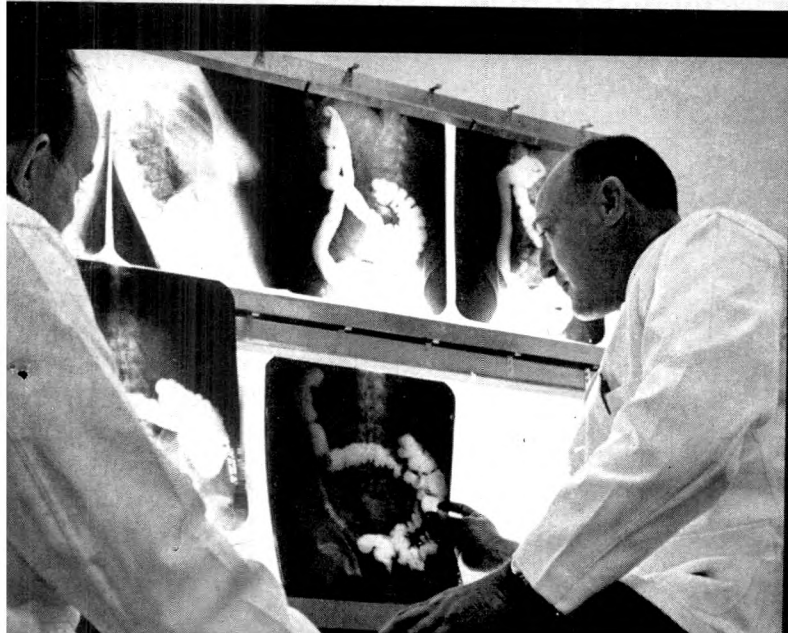
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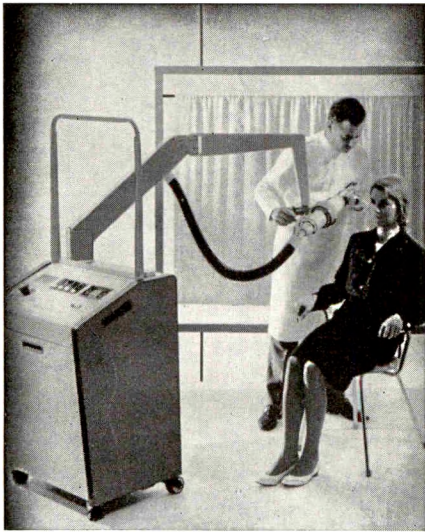
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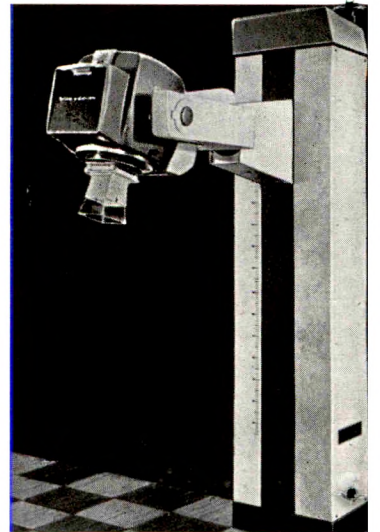
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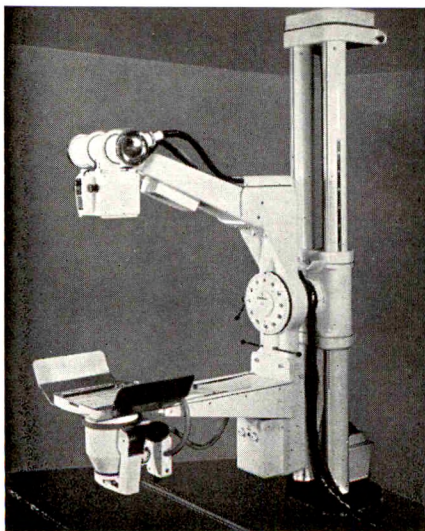
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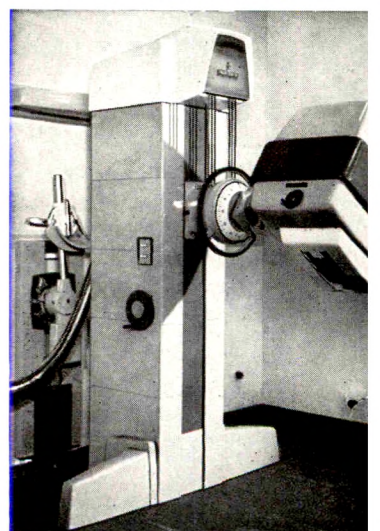


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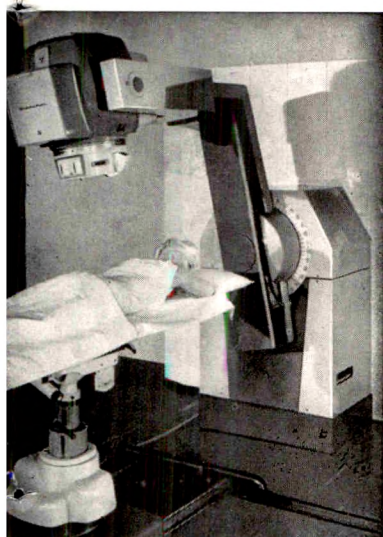
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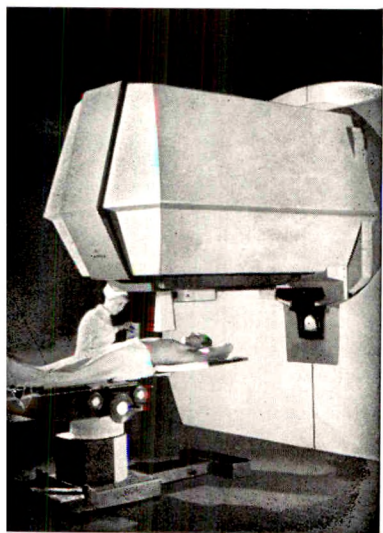
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- **SKULL RADIOGRAPHY: A Simplified System** by Joseph Selman, *University of Texas, Tyler*. Sept. '66, about 194 pp. (7 × 10), about 205 il.
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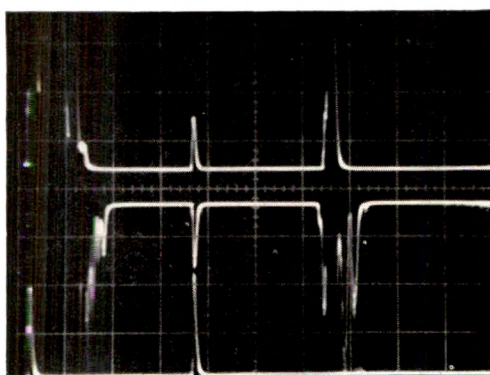
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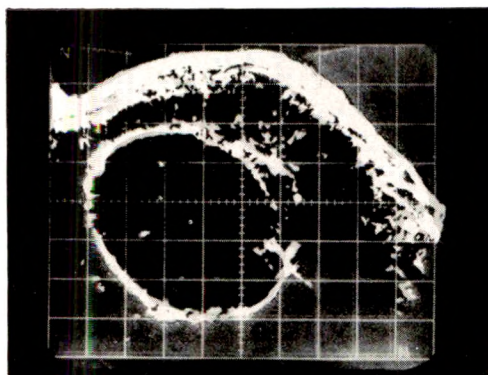
# the diagnostic duality of ultrasound

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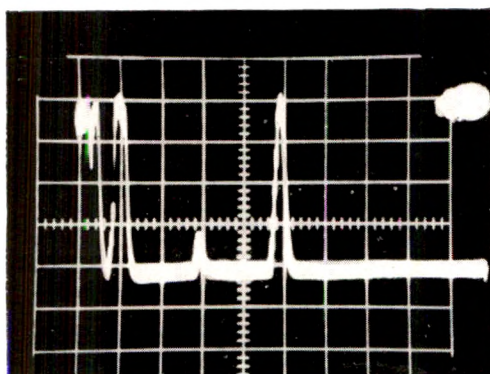


*Location of Brain Midline*

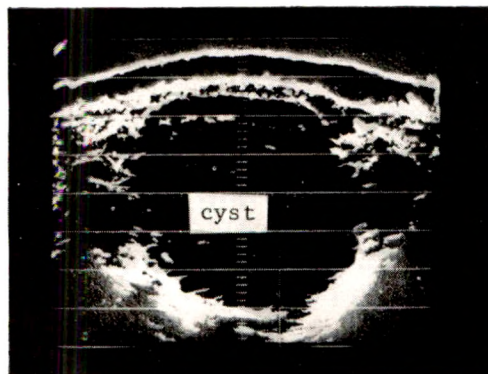
## VISUALIZATION ("B" scan)



*Sagittal section of Uterus demonstrating Fetal Head.*



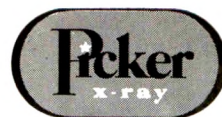
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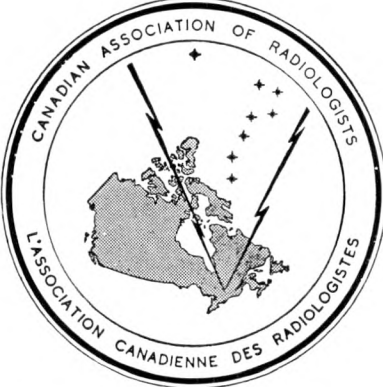
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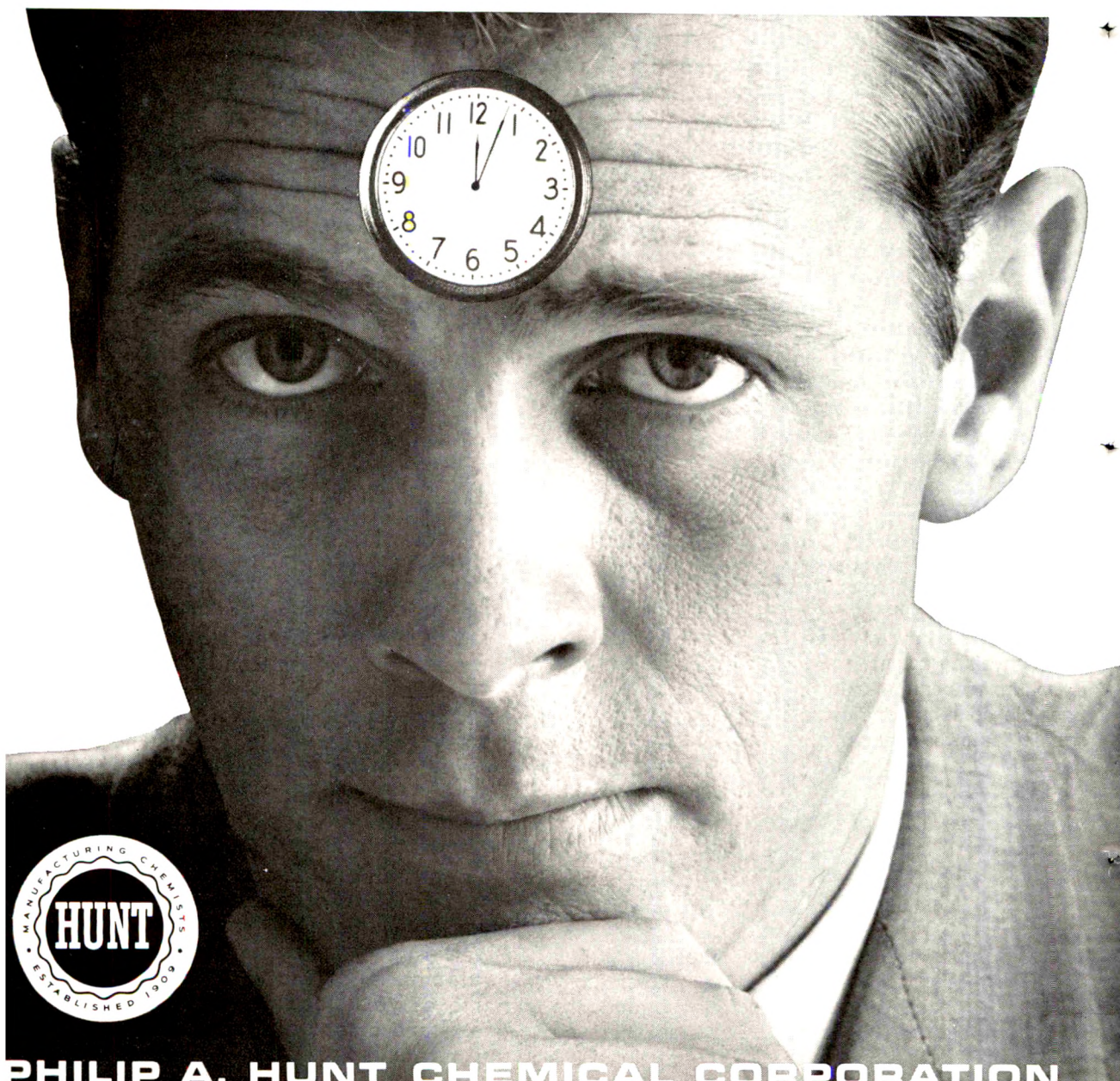
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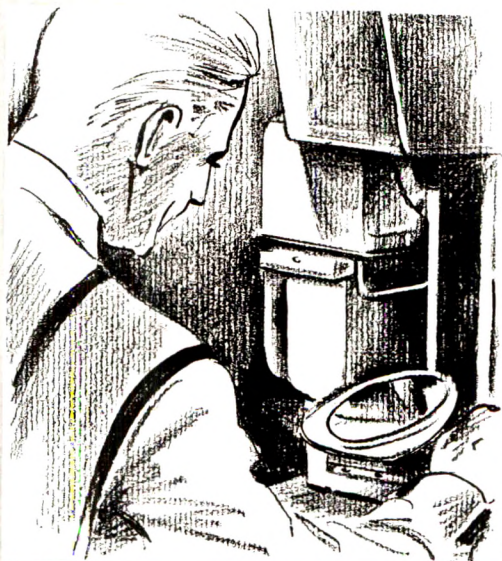
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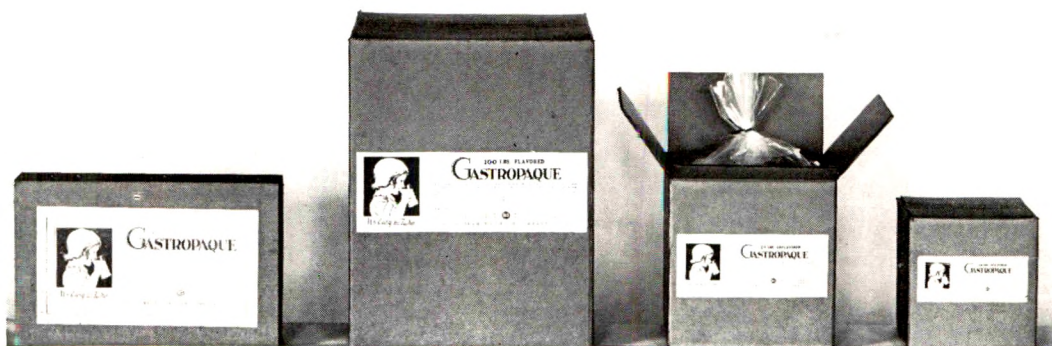
Applied Radiation Corporation .....	xliv, xlv
Atomic Energy of Canada Ltd. ....	xiii
Automatic Seriograph (Div. of Litton Industries) .....	viii
Barber-Coleman Company .....	xi
Barnes-Hind Barium Products .....	liii
Bell-Craig, Inc. ....	i
Buck X-Ray Company .....	lxi
Continental X-Ray Corporation .....	lix
du Pont, E. I., de Nemours Co. ....	xvi, xvii
Eastman Kodak Company .....	xlvi, xlvii
General Aniline & Film Corporation .....	xxxviii, xxxix
General Electric Company .....	xxix, xxx, xxxvii
Gray Pharmaceutical Company .....	xxxii
Halsey X-Ray Products Company .....	xii
Hunt, Philip A., Chemical Corporation .....	lviii
Ilford, Inc. ....	xxxv
Keleket X-Ray Corporation .....	xxxvi
Liebel-Flarsheim Company (Div. of Ritter Pfaudler Corporation) .....	x
Lippincott, J. B., Company .....	xli
Low X-Ray Corporation .....	ix
Machlett Laboratories, Inc. ....	xix, xx
Mallinkrodt Chemical Works .....	xiv, xv
Metrix, Inc. ....	liv
North American Philips Company .....	xxxii, xxxiii
Nuclear-Chicago Corporation .....	xviii
Nuclear Consultants Corporation .....	xxxiv
Ortho Pharmaceutical Corporation .....	xxi
Pako Corporation .....	li
Pickar X-Ray Corporation .....	Second Cover, xxviii, iv, Fourth Cover
Profexray, Inc. ....	xxiv, xxv
Pyne X-Ray Corporation .....	vii
Schick X-Ray Company .....	vi
Schneeman Electronics, Inc. ....	lvi
Siemens Medical of America, Inc. ....	i, li
Squibb, E. R., & Sons .....	v, lxii
Standard X-Ray Company .....	lvii
Thomas, Charles C, Publisher .....	liv
Varian Associates .....	xlvi, xlvii
Visi-Shelf Files, Inc. ....	xi
Westinghouse Electric Corporation .....	xxvi, xxvii
Winthrop Laboratories .....	xxii, xxiii, xlii, xliii
Wolf X-Ray Corporation .....	Third Cover

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**"...[pulmonary embolism] may exist in a grave form for a considerable time without objective signs being present on physical examination or routine chest films."<sup>1</sup>**

Pulmonary embolism is a mimic. Because its symptoms resemble those of other cardiorespiratory diseases—particularly myocardial infarction<sup>2,3</sup> and pneumonia<sup>4</sup>—investigators have long sought simple and certain methods of diagnosing it.

any diagnostic clues—but often no clinical picture

Until a few years ago diagnosis depended primarily on the clinical history, physical findings, chest films, electrocardiograms, angiography, and pulmonary function studies. Each of these is helpful. Sometimes not even *all* of them were conclusive.

Surgery, prolonged immobilization, metastatic carcinoma and trauma often precede pulmonary embolism—but are not necessarily followed by it. Pain, dyspnea, hemoptysis may signal pulmonary embolism—but they aren't necessarily peculiar to it. The electrocardiogram may be normal in spite of it.<sup>5</sup> And there is no pathognomonic radiographic picture of pulmonary embolism.<sup>5</sup>

It is clinically valuable, however, a new diagnostic test should meet two criteria:

it should be correlated with known pathology; i.e., it should be carefully compared with other diagnostic procedures; it should offer information not attainable as easily or as safely as accepted tests.<sup>6</sup>

Lung scintiscanning meets the first criterion and is a most valuable diagnostic tool. It is, however, a time-consuming procedure and one that requires experienced personnel.<sup>2</sup>

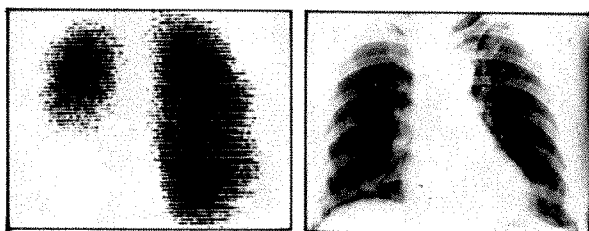
Lung scintiscanning can detect an obstruction of the pulmonary circulation as soon as it is established.<sup>7</sup>

It is not only the diagnostic procedure of lung scanning both safe and easy,<sup>2</sup> but there is information that is not attainable on chest films. "...it appears that the lung scan can point to the site of the embolic lesions before signs of lung infarction are recognizable on plain chest films."<sup>1</sup>

The lung scan and the x-ray shown confirm this statement.\* The lung scan of this female patient, aged 58, was taken August 13, 1965 with Albumotope-LS (Squibb Aggregated Radio-iodinated [<sup>131</sup>I] Albumin [Human]). Pulmonary emboli are clearly evident. The x-ray, taken the same day, shows no radiographic evidence of pulmonary emboli.

Lung scanning meets both criteria for a clinically valuable diagnostic test. Findings are correlated with pulmonary function studies, angiography, pathology and the clinical state of the patient. And, scanning with Albumotope-LS has been proven to be "simple, rapid, and safe in the diagnosis, localization and

illustrations furnished through the courtesy of George V. Taplin, M.D., Harbor General Hospital, Torrance, California.



ultimate fate of pulmonary emboli."<sup>2</sup> But the lung scan should not be relied upon as the only diagnostic procedure in the diagnosis of pulmonary embolism.<sup>1</sup>

**Dosage and Scanning Procedures:** Recommended scan doses of 150 to 300 microcuries of Albumotope-LS (Squibb Aggregated Radio-iodinated [<sup>131</sup>I] Albumin [Human]) depending on the instrumentation available and the techniques employed. Scanning can immediately follow administration of slow intravenous injection or be delayed up to 1 to 1½ hours depending on preferred technique.

**Side Effects and Precautions:** Extensive clinical use of Albumotope-LS has not borne out the hypothetical possibility that particles of large size might induce deleterious cardiovascular or cerebrovascular effects. No antigenic properties have been specifically related to this product; one patient with a known history of angioneurotic edema, who had been given Lugol's solution in conjunction with aggregated radio-albumin similar to Albumotope-LS, developed urticaria.

Radioisotopes should not be used in pregnant women, nursing mothers, or in patients under 18 years of age unless indications are very exceptional.

**Available:** As a sterile, non-pyrogenic, aqueous suspension. Each cc. contains approximately 1 mg. aggregated human serum albumin labeled with 800-1500 microcuries of iodine-131 at time of manufacture. Also contains 0.9% benzyl alcohol as a preservative.

**References:** 1. Haynie, T. R., et al.: J. Nucl. Med. 6:613, 1965. 2. Sabiston, D.C., Jr., and Wagner, H.N.: Ann. Surg. 160:575, 1964. 3. Cooley, R.N., and Donner, M.W.: Am. J. M. Sc. 247:601, 1964. 4. Wagner, H.N., et al.: New England J. Med. 271:377, 1964. 5. Hinshaw, H.C., and Garland, L.H.: Diseases of the Chest, ed. 2, Philadelphia, W. B. Saunders Co., 1963, pp. 438-9. 6. Dworkin, H.J., et al.: Michigan Med. 64:829, 1965. 7. Quinn, J.L., et al.: Radiology 82:315, 1964. 8. Taplin, G.V., et al.: Health Physics 10:1219, 1964.

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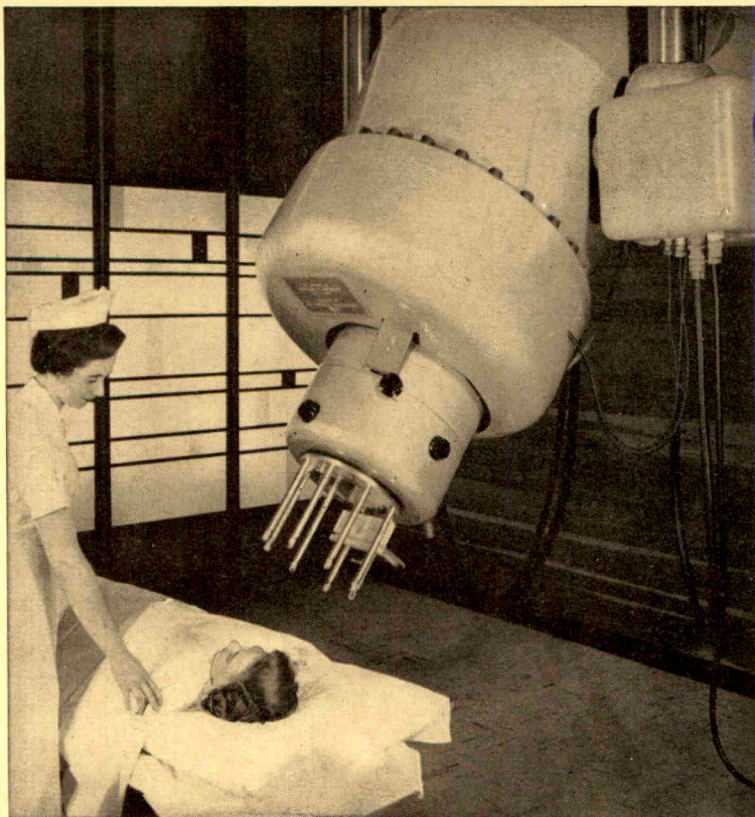
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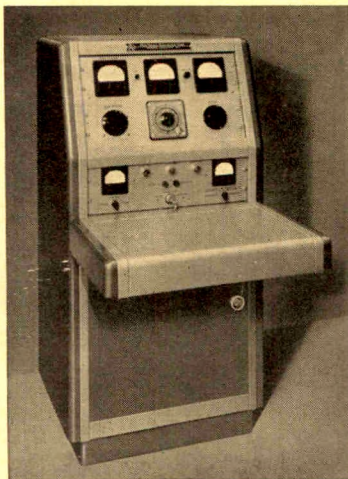


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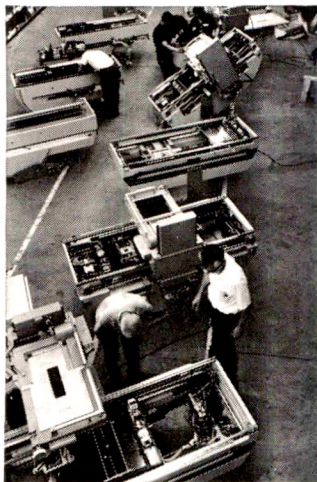


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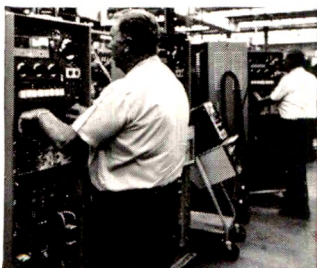
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VOL. 98

DECEMBER, 1966

No. 4

## TABLE OF CONTENTS

<i>Problems in Radiology of Today and the Future; The President's Address.</i> CLYDE A. STEVENSON, M.D. . . . .	777
<i>Supravalvular Aortic Stenosis; Roentgen Analysis of Twenty-Seven Cases.</i> GERALD J. KURLANDER, M.D., EUGENE L. PETRY, M.D., HOOSHANG TAYBI, M.D., PAUL R. LURIE, M.D., AND JOHN A. CAMPBELL, M.D. . . . .	782
<i>Congenital Aortic Stenosis.</i> SHOICHI D. TAKEKAWA, M.D., OWINGS W. KINCAID, M.D., JACK L. TITUS, M.D., AND JAMES W. DUSHANE, M.D. . . . .	800
<i>Supravalvular Aortic Stenosis.</i> EDWARD A. KUPIC, M.D., AND HERBERT L. ABRAMS, M.D. . . . .	822
<i>Catheter Position: An Aid in Diagnosis of Dissecting Aneurysm of the Thoracic Aorta.</i> GLEN G. CRAMER, M.D., AND KURT AMPLATZ, M.D. . . . .	836
<i>Calcified and Dilated Ascending Aorta Due to Atheromatous Occlusive Disease Simulating Coarctation of the Aorta; A Report of a Case and Theory of Pathogenesis.</i> ISRAEL STEINBERG, M.D. . . . .	840
<i>Atherosclerosis in the Rabbit Visualized by Cardioventriculography and Subtraction.</i> CHARLES P. OLINGER, M.D. . . . .	844
<i>Diversion of the Inferior Vena Cava into the Left Atrium Following Atrial Septal Defect Closure.</i> TOM W. STAPLE, M.D., THOMAS B. FERGUSON, M.D., AND BRENT M. PARKER, M.D. . . . .	851
<i>Observations on Pulmonary Embolism and the Pulmonary Angiogram.</i> STEPHEN N. WIENER, M.D., JOSEF EDELSTEIN, M.D., AND BERNARD L. CHARMS, M.D. . . . .	859
<i>Angiocardiographic Diagnosis of Pericardial Effusion.</i> ISRAEL STEINBERG, M.D., AND BERNARD A. HECKMAN, M.D. . . . .	874
<i>Catheter Arteriography in the Evaluation of Abdominal Aortic Aneurysms.</i> PAUL C. KAHN, M.D., AND ALLAN D. CALLOW, M.D. . . . .	879
<i>Cavography Following Plication of the Inferior Vena Cava.</i> JAMES V. BLAZEK, M.D., RICHARD L. CLARK, M.D., AND CHARLES S. HERRON, M.D. . . . .	888

(Continued on page iv)

<i>Arteriography in Circumferential Compression Edema.</i> J. L. CURRY, M.D., AND W. J. HOWLAND, M.D. . . . .	898
<i>Stationary Waves of the Superficial Femoral Arteries; Report of a Case Occurring in a Patient with Rheumatic Heart Disease and Popliteal Arterial Emboli.</i> ISRAEL STEINBERG, M.D. . . . .	901
<i>Fibromuscular Hyperplasia; An Arterial Dysplasia of Increasing Clinical Importance.</i> A. J. PALUBINSKAS, M.D., DOROTHEE PERLOFF, M.D., AND THOMAS H. NEWTON, M.D. . . . .	907
<i>Blood Pressure Changes Induced by Subclavian Injection of Methylglucamine Diatrizoate (Renografin).</i> JYOJI HANDA, M.D., JOHN S. MEYER, M.D., AND KO SAKAMOTO, M.D. . . . .	914
<i>The Osmotic Effects of Urografin 76 Per Cent and Isopaque 60 Per Cent in Angiocardiography.</i> LARS BJÖRK . . . . .	922
<i>The Effects of Angiotensin on Renal Vascularity in Dogs.</i> MILTON ELKIN, M.D., AND CHIEN-HSING MENG, M.D. . . . .	927
<i>The Value of Nephrotomography in the Differential Diagnosis of Abdominal Masses.</i> RICHARD D. KITTREDGE, M.D., VIRGINIA KANICK, M.D., AND NATHANIEL FINBY, M.D. . . . .	935
<i>An Improved, Simple Technique of Lymphangiography.</i> DR. T. DE ROO . . . .	948
<i>Improved Technique of Lymphangiography.</i> BAO-SHAN JING, M.D. . . . .	952
<i>Safety Guidespring for Percutaneous Cardiovascular Catheterization.</i> CHARLES T. DOTTER, M.D., MELVIN P. JUDKINS, M.D., AND LOUIS H. FRISCHE, M.D. . . . .	957
<i>Photographic Spot Films and Radiographic Spot Films.</i> BERTRAM R. GIRDANY, M.D., STEVEN E. ROSS, M.D., AND FRED A. LEE, M.D. . . . .	961
<i>Investigations in Colored Radiography.</i> HARVEY R. PRINS, D.ENG., J. LAWRENCE KATZ, PH.D., AND FRED W. BILLMEYER, JR., PH.D. . . . .	966
<i>Officers</i> . . . . .	979
<i>Editorials</i>	
<i>The Sixty-seventh Annual Meeting of the American Roentgen Ray Society.</i> TRAIAN LEUCUTIA, M.D. . . . .	980
<i>Douglas A. Quick, 1891-1966.</i> J. H. MAUS, M.D. . . . .	984
<i>The Scientific Exhibits</i> . . . . .	987
<i>The Technical Exhibits</i> . . . . .	1000
<i>News Items</i> . . . . .	1002
<i>Book Review</i> . . . . .	1005
<i>Books Received</i> . . . . .	1005
<i>Society Proceedings</i> . . . . .	1007
<i>Abstracts of Radiological Literature.</i> . . . .	1012
<i>Subject Index to Volume 98.</i> . . . .	1032
<i>Author Index to Volume 98.</i> . . . .	1041



## same day re-examination in oral cholecystography

### obviously practical, but... diagnostically valuable?

UNSATISFACTORY opacification after routine cholecystography doesn't necessarily mean the gallbladder is diseased. Poor absorption of the contrast agent from the gastrointestinal tract, or the inability of the liver to conjugate and excrete it, might just as well be the cause.<sup>1</sup>

#### 'Nonopacification' is not a condition you can treat

To determine the cause of nonopacification, most physicians request re-examination. There are two ways this has been done by the oral route in the past:

- by starting all over again at a later date — and sometimes doubling the dose of contrast agent on the second try;
- by administering more contrast agent the evening of the first unsuccessful examination (and, again, sometimes doubling the dose) and repeating the study the following day.

Both these methods share two inherent disadvantages. They take a good deal of time, and they require the patient to take a good deal of oral contrast agent. The amount given assumes clinical importance because it has been reported higher doses may result in considerable patient discomfort because of diarrhea and other significant side effects.<sup>1</sup>

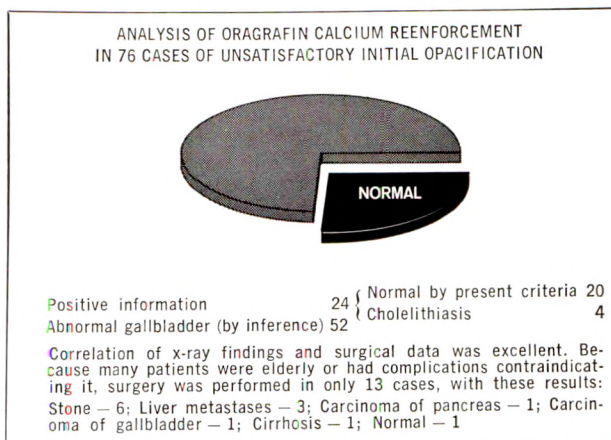
Recently it's been demonstrated, however, that the use of Oragrafin Calcium (Squibb Calcium Iodate) permits repeat films with significant cuts in both time and total dosage.

#### Oragrafin Calcium—absorbed so rapidly you can re-examine the same day

"The advantage of the calcium iodate method is that the examination can be completed in five additional hours with a limited dose of contrast agent."<sup>1</sup>

The method is as productive as it is simple. If the initial opacification is unsatisfactory, the patient is given 3 Gm.

of calcium iodate. Films are repeated 5 hours later. Here the results of a recently published study on same day examination using Oragrafin Calcium:<sup>1</sup>



Had the initial failure of opacification been assumed to be due to gallbladder disease, the 20 patients (26%) found normal by re-examination might have been erroneously diagnosed and might have undergone unnecessary surgery.

"Same day re-enforcement oral cholecystography utilizing calcium iodate . . . is a rapid, accurate, low-dose method obtaining additional information about a gallbladder which is unsatisfactorily opacified following routine cholecystography."<sup>1</sup>

There are other advantages to such rapid absorption. Even without prior preparation of the patient, both cholangiography and cholecystography can be performed on the same day. Optimal concentration in the hepatic and biliary ducts usually occurs within 1 to 3 hours. Although the gallbladder is optimally opacified approximately 10 hours after ingestion of Oragrafin, diagnostically valuable information can often be obtained within 5 hours or less.

**Dosage and Administration:** For oral combined cholangiography and cholecystography: *Same day* — Oragrafin Calcium Granules — 2 packets 1 hour before initiating examination. *Overnight* — Oragrafin Calcium Granules — 1 packet or Oragrafin Sodium Capsules (Squibb Sodium Iodate) — 6 capsules 10 to 12 hours before radiography, plus an additional packet of Granules one hour before examination.

**Supply:** Oragrafin Calcium Granules — single-dose foil packets of 3 Gm. of calcium iodate granules, providing 61.7% iodine content. Oragrafin Sodium Capsules — 0.5 Gm. sodium iodate per capsule, providing 61.7% iodine content.

**Contraindications:** Contraindicated for persons sensitive to oral iodine compounds or for patients with combined renal and hepatic disease, severe kidney impairment. Gastrointestinal disorders (which may result in inadequate biliary secretion of medium) often impair visualization.

**Side Effects and Precautions:** Both calcium and sodium iodate are usually well tolerated. Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium Granules and restricting the dosage to 3 Gm.

Hypersensitivity reactions may include urticaria, serum sickness reactions (fever, rash, arthralgia), other skin reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of hypersensitivity diathesis. Antihistamines and corticosteroids are used to control these side effects, but the occasional serious anaphylactoid reaction requires the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids. For full information see Package Insert.

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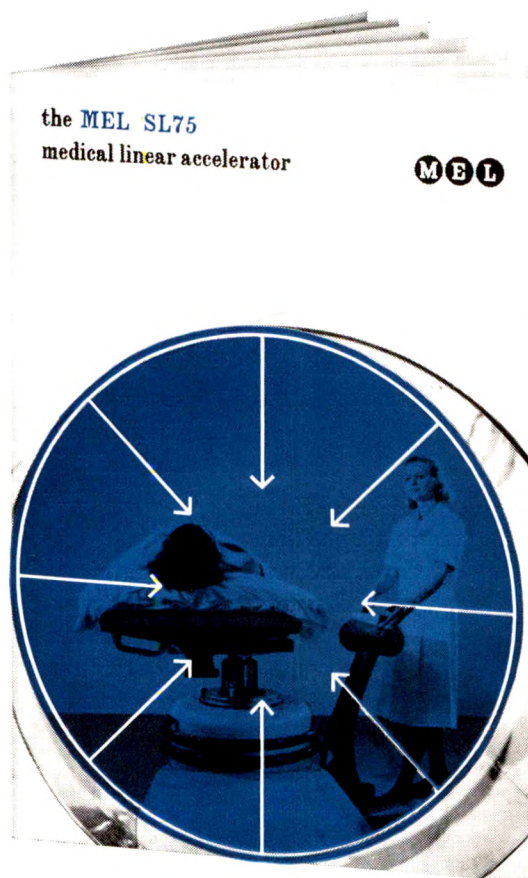
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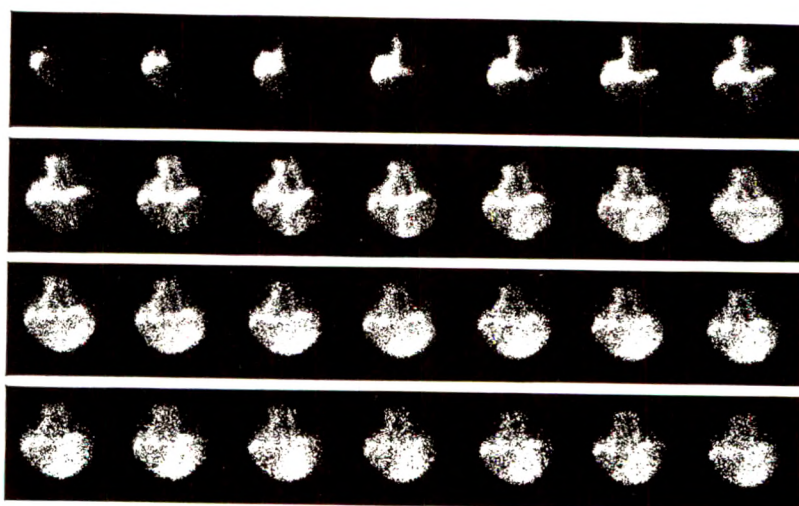
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is making important contributions  
to modern diagnostic procedures.  
Get the full story from your local  
Nuclear-Chicago sales engineer.  
Or write directly to us.**

You'll receive detailed information  
on Pho/Gamma's clinical applica-  
tions and on new Pho/Gamma III.

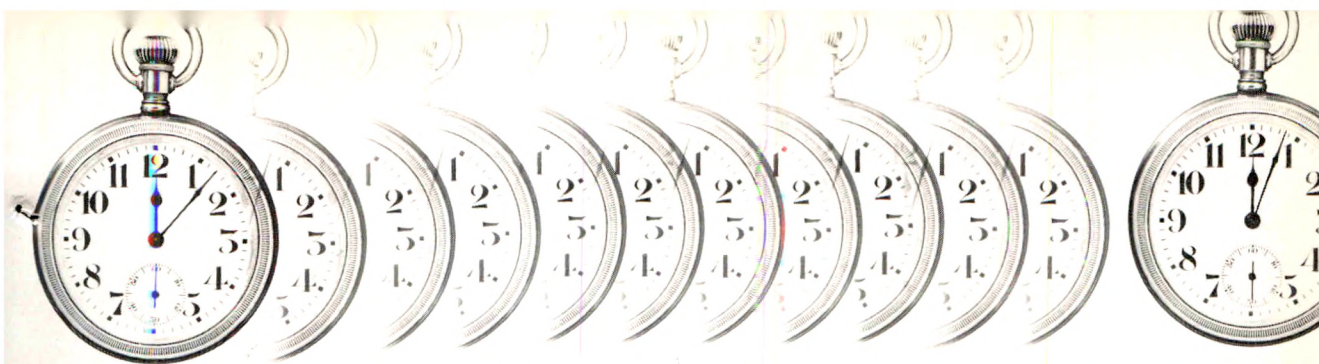
NUC D-8-208



**NUCLEAR-CHICAGO  
CORPORATION**

307 East Howard Avenue, Des Plaines, Illinois 60018, U.S.A. / Donker Curtiusstraat 7, Amsterdam W, The Netherlands.





Going from conventional to new 3½ minute processing



# TAKE ALONG LIQUAMAT

(it goes great at either speed)

It's no secret that many x-ray departments are switching from conventional to 3½ minute processing. Inevitably questions arise about what chemistry to use. And the variety being offered is pretty confusing.

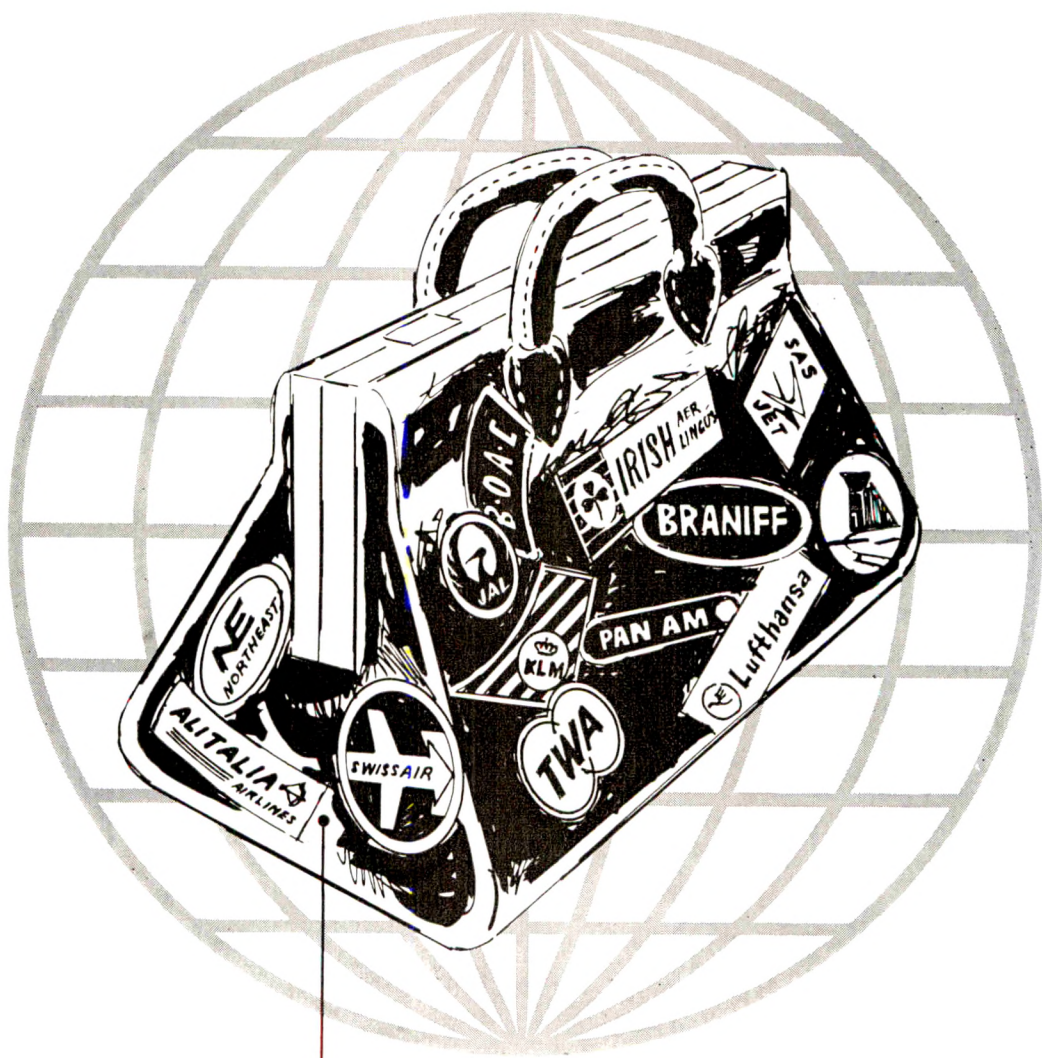
Relax. GAF Liquamat Red and Liquamat Clear work just as well at 3½ minutes as at standard speeds. So conversion is possible without losing the hospital-proved

Liquamat benefits: superb image... clean-working characteristics... color-coded containers... long working life... the red color-indicator in Red Liquamat that shows changes in the solution... the light, one-gallon bottles handle with ease.

So when you go to 3½ minute processing, take along Liquamat. You'll have more going for you.



GENERAL ANILINE & FILM CORP.



**DO YOU      NEED SPECIALIZED  
EQUIPMENT, DOCTOR?**

Our business is to provide high  
quality specialized equipment  
—and service to match—in cooperation  
with your local x-ray dealer.



**SCHICK X-RAY CO., INC.**

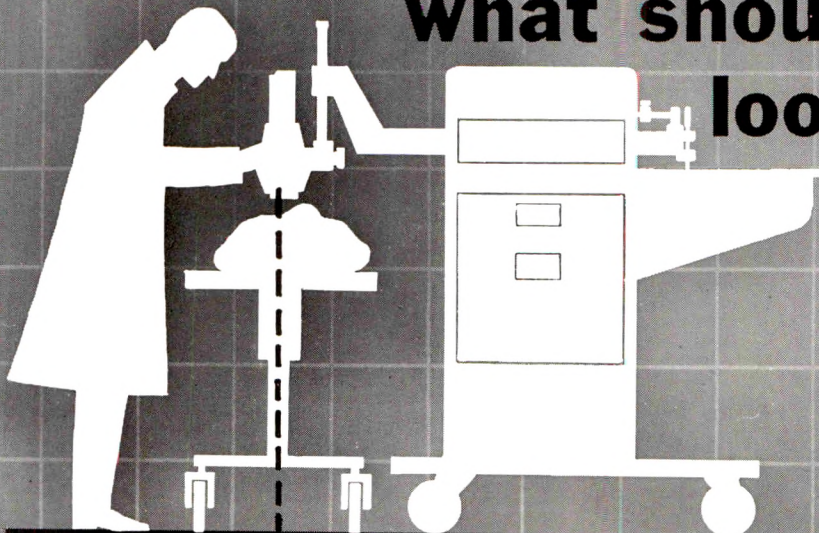
444 North Lake Shore Drive

Chicago, Illinois 60611



when investing in a scintillation scanner

what should you  
look for?



among other things...

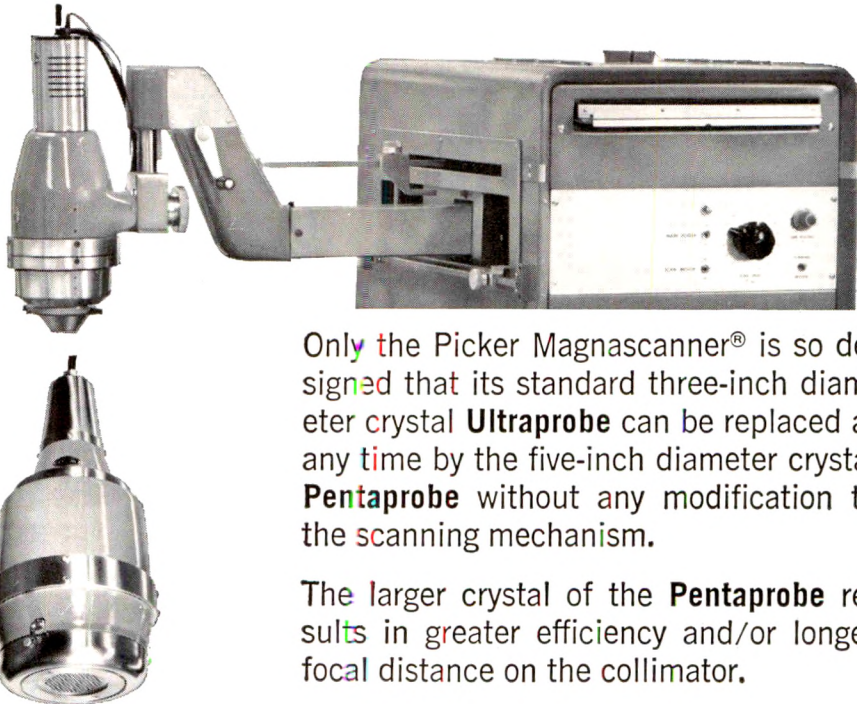
**the ability to upgrade**

from this

3"

to this

5"



Only the Picker Magnascanner® is so designed that its standard three-inch diameter crystal **Ultraprobe** can be replaced at any time by the five-inch diameter crystal **Pentaprobe** without any modification to the scanning mechanism.

The larger crystal of the **Pentaprobe** results in greater efficiency and/or longer focal distance on the collimator.

**PICKER**  
nuclear

**Magna Scanner®**

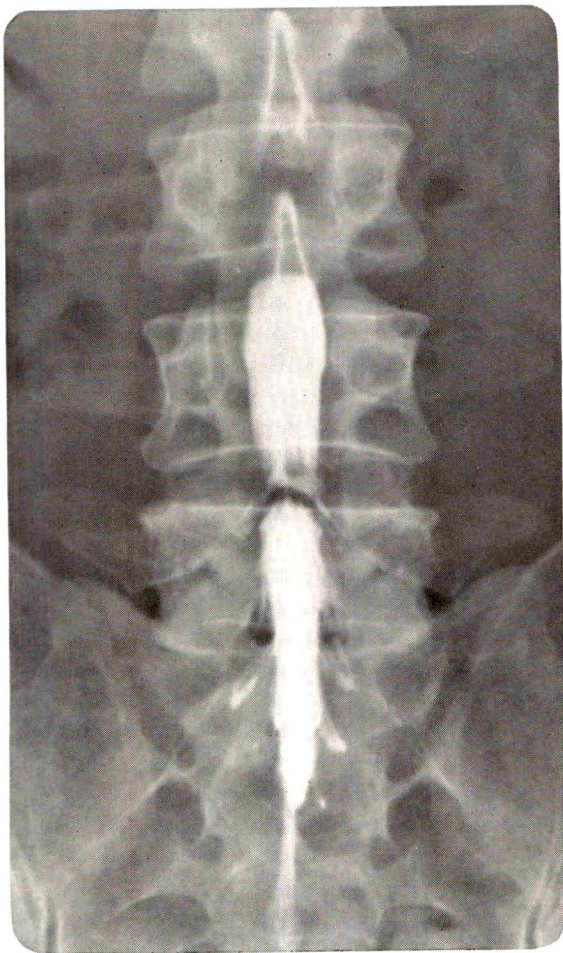
the **versatile** scanner / the **proven** scanner

PICKER NUCLEAR  
DIVISION

PICKER X-RAY CORPORATION  
WHITE PLAINS, NEW YORK



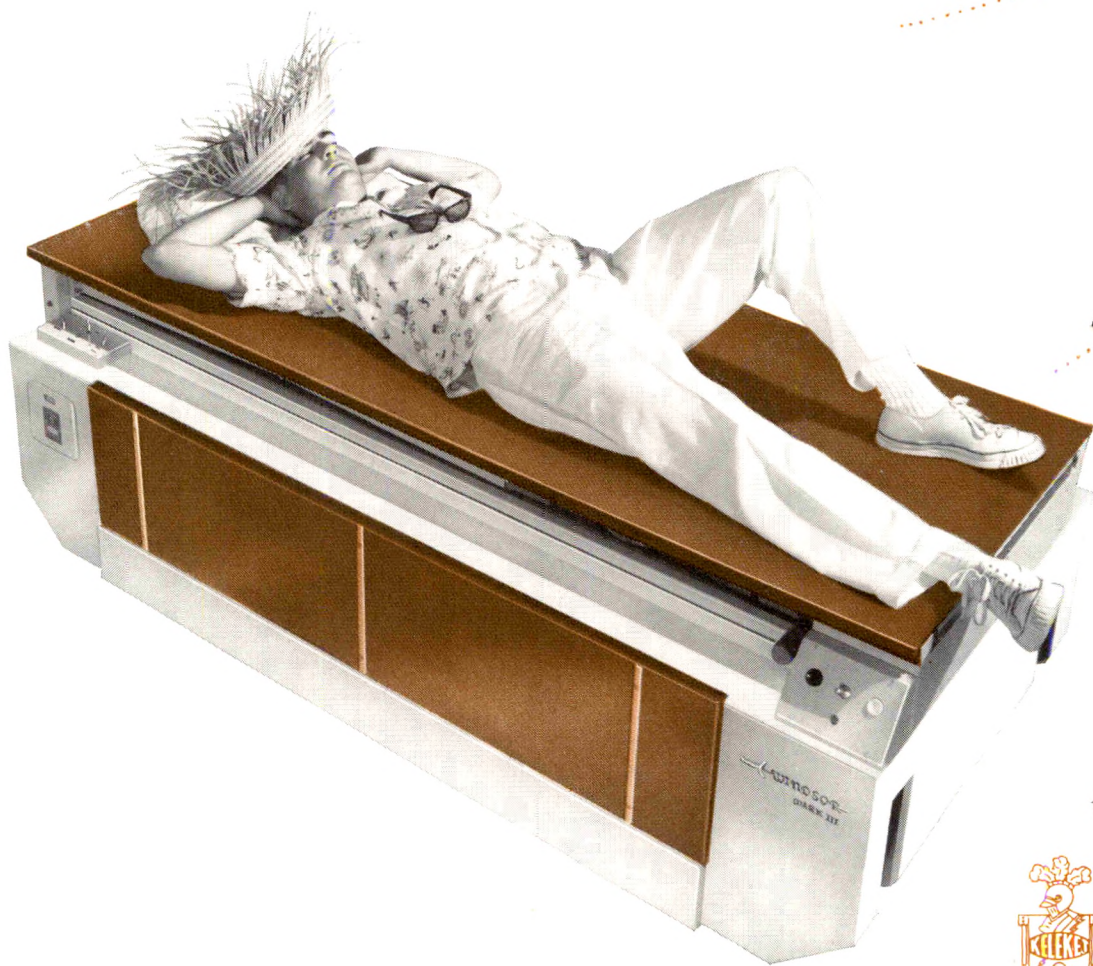




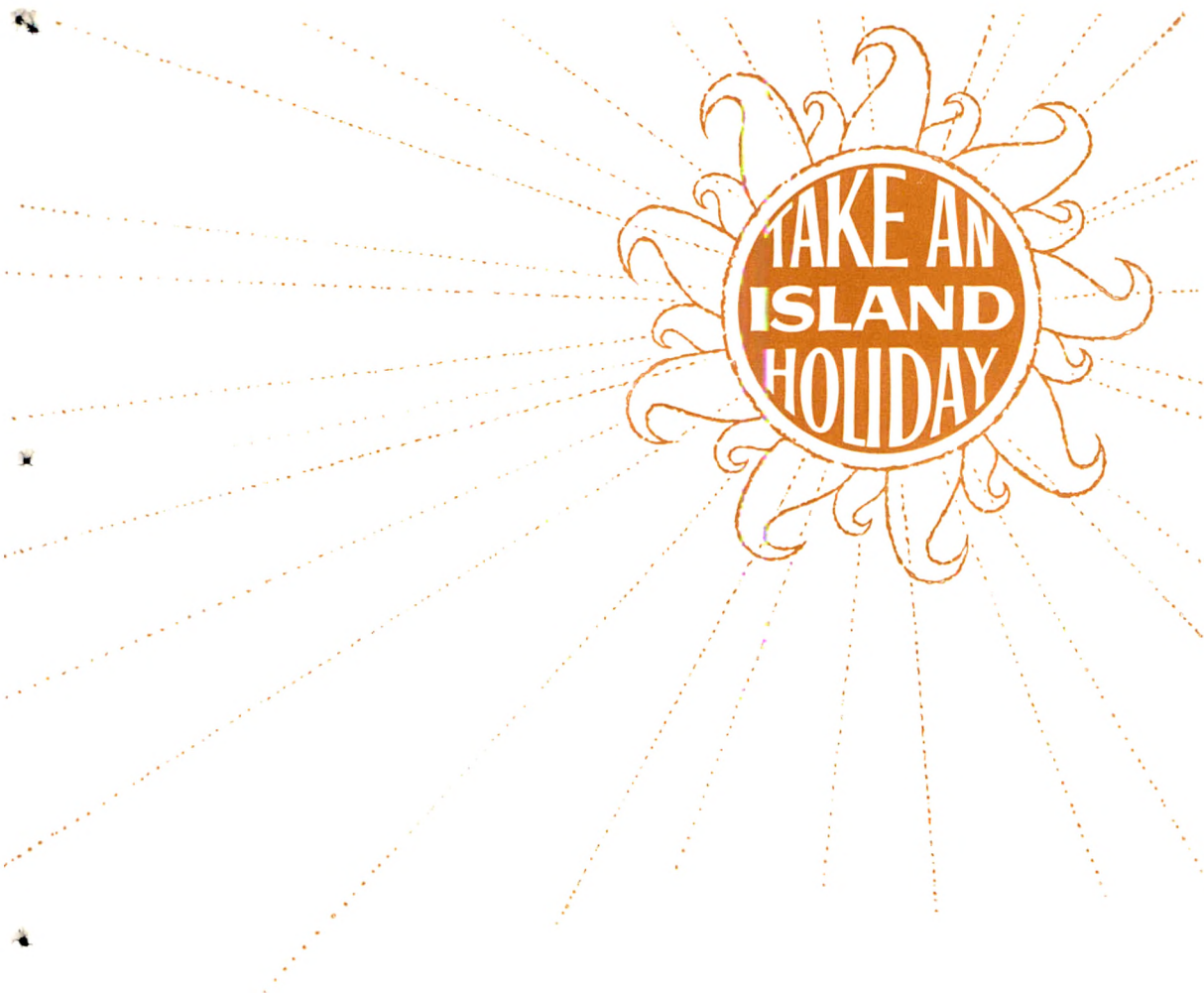
The patient, a 46-year-old male, had persistent low back pain radiating down both lower extremities posteriorly, and numbness and pain over both heels. X-ray examinations of back and hips, made 18 months before, following an automobile accident, were reported as normal.

Myelographic examination using PANTOPAQUE [Iophendylate Injection] showed a large defect at the fourth lumbar interspace, which was interpreted to be due to discal herniation in a narrow canal. At surgery, a large herniated intervertebral disc in a very narrow lumbar vertebral canal was found and removed.

*“PANTOPAQUE” is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized in the laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iophendylate Injection, U.S.P., by Lafayette Pharmacal Inc. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*







**Accessibility:** Nothing is quite as easy to get to, get on, get around as an island X-ray table.

**Visibility:** All around viewing across the board, from either side of the table.

**Freedom from fatigue:** Spot film tower and tunnel assembly are fully counterbalanced, easily swung into play or parked out of the way for fatigue-free fluoroscopy.

**Summing up:** Only island units give you this much freedom, this ease of operation, this range of functions, this holiday from the chores associated with any other type of X-ray table. And your only source for true island table design is Keleket, for 65 years, the first name in medical X-ray. Ask for Windsor island tables.



**KELEKET**

A Division of Laboratory For Electronics, Inc.

WALTHAM, MASSACHUSETTS 02154

Medical X-ray Equipment • Accessories and Supplies

**Du Pont  
focuses  
its science  
and energy  
on  
Diagnostic  
Clarity**





We're interested in one thing when our film is snapped on your viewbox: how well you see what you want to see.

Your ability to read, to interpret, to satisfy highly-trained insights; this is the measure we go by.

And so we apply our multi-faceted science to that end.

We developed CRONAR®—a thinner, clearer, crisper polyester film base to improve radiographic quality. It also happened to revolutionize automatic processing.

We built the world's largest, most modern plant devoted exclusively to x-ray film manufacture. It was designed to accommodate a breakthrough in emulsion technology. It also gave us important advances in quality control.

We matched three new screens to our CRONEX® films to provide combinations for your most advanced techniques. Not surprisingly, they offer the widest choice of contrast and speeds available.

Not all the improvements in film are coming from Du Pont. But most are—and will.

And you can be certain that each new Du Pont advance will continue to focus primarily on Diagnostic Clarity.

With, quite probably, continued side benefits.

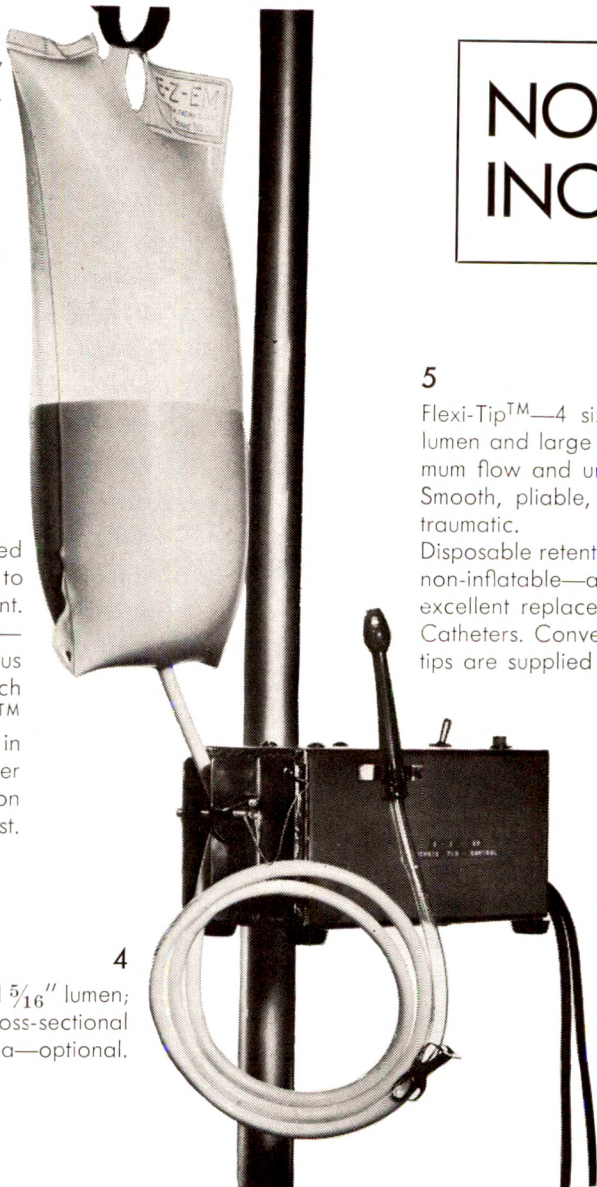
## CRONEX X-ray Products



Better Things for Better Living  
... through Chemistry

# The E-Z-EM® System is more than just Barium and a bag.

The complete E-Z-Em Barium enema system is guaranteed to help you increase your patient load with maximum diagnostic reliability, safety and economy. E-Z-Em has been used in over 1,500,000 procedures. Your suggestions have guided us to 6 new improved features which make E-Z-Em the most practical B. E. system available.



1 Standard capacity now 3000 cc; 4000 cc optional.

2 New pleasant smelling, stronger, vinyl formulation.

3 Prepackaged with improved fast-mixing Bari-Em™ to the exact weight you want. Colloidal particle size—under one micron—plus positive charge on each particle keeps Bari-Em™ stable—in the bag and in the patient. Any other leading formulation packaged on request.

4 Kink-free tubing with full  $\frac{5}{16}$ " lumen;  $\frac{3}{8}$ " lumen—37% more cross-sectional area—optional.

5 Flexi-Tip™—4 sizes—all with large lumen and large side holes for maximum flow and unobstructed drainage. Smooth, pliable, comfortable, non-traumatic. Disposable retention tips—inflatable and non-inflatable—available. Both are excellent replacements for Foley Catheters. Conventional rigid plastic tips are supplied as standard.

6 Remote Flo-Control™—gives complete control of barium flow rate to you. As good as an extra pair of hands or another technician. Supplied with either foot or hand switch.

NO PRICE INCREASE

E-Z-Em® is now stocked by most X-Ray dealers. Ask your salesman for a sample and demonstration. If he doesn't have E-Z-Em, ask him to get samples for you, or write direct:

**E-Z-EM**

**"THE SYSTEM OF CHOICE"**

Patent No. 3,177,871

32 South Street,  
Port Washington, L. I., N. Y.,  
516-PO 7-4743

## Bibliography

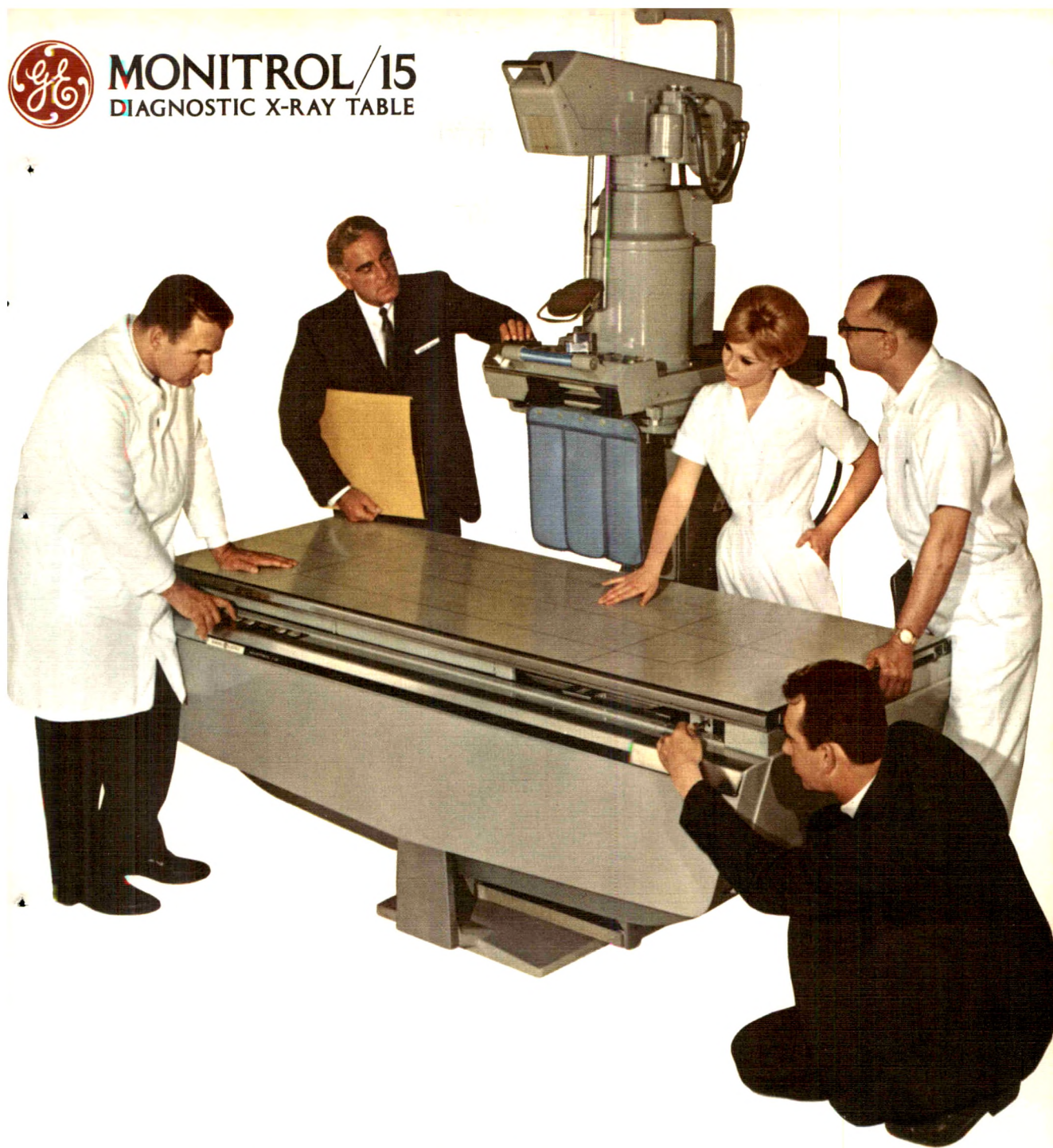
1. Meyers, P. H.: J.A.M.A. 173: 1589, August 6, 1960. 2. Nathan, M. H.: Am. J. Roentgenol., Rad. Ther. and Nuclear Med. 81: 650, 1959. 3. Steinbach, H. L., et al.: J.A.M.A. 174: 1207, Oct. 29, 1960. 4. Pochaczewsky, R., et al: Radiology 77: 831, (Nov.) 1961.

**Ask about Sol-O-Pake™,  
the pleasant tasting barium formulation for upper GI's.**





## MONITROL/15 DIAGNOSTIC X-RAY TABLE



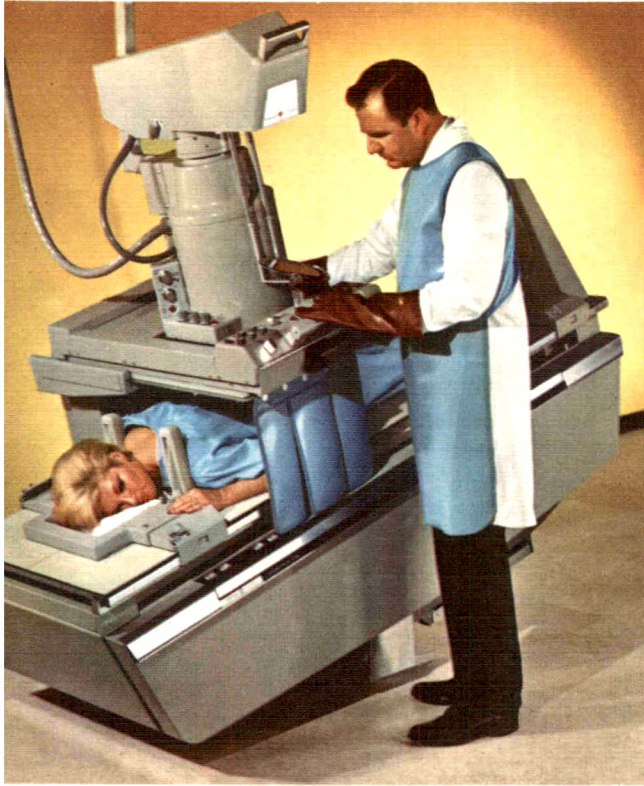
### ...invites critical inspection

**Monitrol/15**—that's the new name to remember when you're selecting a heavy-duty diagnostic x-ray table. Designed and built to perform all radiographic and fluoroscopic functions, this new 90-15° table contains operational features never offered before, by anyone. A true, uncluttered island design, speeded-up powered motions and true field collimation of the fluoroscopic beam are but a few. And General Electric has produced all this at a moderate price.



# MONITROL/15

**The New 90-15° Table Built For Heavy-Duty Use,**



The new Monitrol/15 fills the void between light-weight 90-15° tables and heavy-duty 90-90° hospital tables. It's built to take around-the-clock use and is offered by General Electric at an easy-to-take price.

Every newly-designed feature of the Monitrol/15 is aimed at easing the task of the radiologist. For instance—the fluoroscopic tower is lower and far lighter in weight to let you move it with minimum effort and park it faster. Result: a less fatigued radiologist at the end of every examination. Glide-back parking of the spot film device is standard and allows for easy patient positioning and fast table top clearance for radiography.

The Monitrol/15 accommodates the entire Fluoricon line of image intensifiers. The fluoroscopic beam is defined by the G-E True Field Collimator for improved quality spot film and fluoroscopic image. Collimator is controlled by a "servo-drive system" providing an automatic relationship between blade and control handle positions. Feature for feature, the Monitrol/15 offers more in patient comfort and operator convenience than any diagnostic table made today. It's a proud addition to the General Electric line of superior x-ray equipment.

*Progress Is Our Most Important Product*



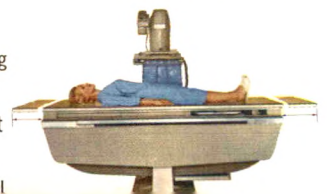
## **Radiologist-Designed Features for Total Diagnostic Performance**

**Spot Filming**—Monitrol/15 accepts a choice of top loading, end loading spot film devices and the Fluoricon Pedestal. New low tower provides for spot film travel of 11-in. Distance from table top to underside of spot film device ranges 7-in. to 18-in.



**Controls**—Dual control is standard on the Monitrol/15. Controls are positioned for ease of access on both the table-front ledge and on the spot film device. The table-mounted switches are barium-proof.

**Power Positioning**—The moving table top with independent centering switch provides for a choice of table head-and-foot travel at installation—choose from 30-in. head/30-in. foot, 45-in. head or 45-in. foot travel





# A NEW VIEWER FOR RADIOLOGISTS WHO WOULD RATHER DIAGNOSE FILMS THAN HANDLE THEM

Meet the Fluoroline mobile magazine multiple film viewer system — a new viewer that brightens and lightens film interpretation and conserves valuable time for the busy radiologist.

That long name — mobile magazine multiple film viewer system — really explains the full benefit of this new viewer. Each removable magazine of 10 display panels holds up to 80-14"x17" radiographs (280-8"x10" or 440-5"x7" films) for rapid reference on the eight bank (4 over 4) crystal clear, uniformly bright illuminator. Technicians simply load a series of radiographs into the magazine and roll it to the viewer. Once

locked into place, film interpretation proceeds smoothly without the need of the radiologist ever touching a film.

Those valuable radiographs are protected by the smoothly operating Fluoroline viewer. The lightweight panels are counterbalanced for easy positioning and the motor-driven illuminator banks contain automatic safety stops to prevent panel or film damage.

There's much more to know about the functional free-standing or wall-installed Fluoroline viewers. For complete details call your G-E x-ray representative or write X-Ray Department, General Electric Co., Milwaukee, Wisconsin 53201. Room A-121.

*Progress Is Our Most Important Product*

GENERAL  ELECTRIC

# FLUOROLINE®

## MOBILE MAGAZINE FILM VIEWER



# These X-ray generators were designed for radiologists.

(And patients. And technicians.  
And purchasing agents. And service personnel.)

The trick in designing x-ray generators is to remember that it's much more than just an exercise in meeting engineering specifications and then housing the final result in handsome cabinetry. X-ray generators come in contact with a lot of people whose needs—as people—deserve consideration. "User orientation" this is now being called. And we practiced it before anyone had a name for it.

In practical specific terms, what can be said about the new Galaxy generators that will make this claim believable? Take compensation for line voltage variations. With these Galaxy generators you literally can not omit this act because compensation for line voltage variations occurs automatically when you select the kilovoltage you'll use. You don't have to make a separate line voltage adjustment. You can't forget it. You can't bypass it. (Incidentally, stabilization during exposure is accomplished by a "millistabilizer" which automatically adjusts for things like temperature variations, space charge or filament evaporation in the x-ray tube.)

The implications of all this: operation is easy, results consistent.

Other examples: We have always paid particular attention to the positioning of the controls and meters that the operator must use and read—as technicians will tell you. They'll be happy with the judi-

cious use of push buttons to shorten setup time and reduce mistakes.

Service People (and, hence, users) will like these generators because: this is an exceptionally reliable design which minimizes service in the first place; we make our own components whenever what the market offers is not exactly what we need. When service is needed, front access to circuitry eliminates pulling the equipment away from the wall and into the work areas. And, finally, the fact that we have factory-trained servicemen almost everywhere doesn't hurt a bit in helping to minimize human frustrations.

For additional information about Galaxy generators write for literature.

Picker X-Ray Corporation,  
White Plains, New York





# MODERNIZE your X-ray equipment



with  
these  
NEW

# DUNLEE

## SDV RECTIFIERS

### Maximum Peak Inverse Ratings

SDV-100	100 KvP
SDV-125	125 KvP
SDV-150	150 KvP

### Maximum Average Load Current

1/10 sec.	1000 Ma
2 sec.	600 Ma
Continuous	100 Ma

*Consult your x-ray  
Dealer or write*

These Dunlee SDV rectifiers are hermetically sealed high voltage silicon units designed for use in your present x-ray generators. They can be used as direct replacements of the hot cathode vacuum rectifiers . . . they provide longer life, in most cases as long as the original x-ray equipment lasts . . . they operate with greater efficiency and low forward drop . . . and they are higher in mechanical strength. Simple and safer to operate, these silicon rectifier cartridges are available in new units from the manufacturer of your equipment . . . just ask for them when you buy your next x-ray unit. Use as replacement for your present valve tubes as well.

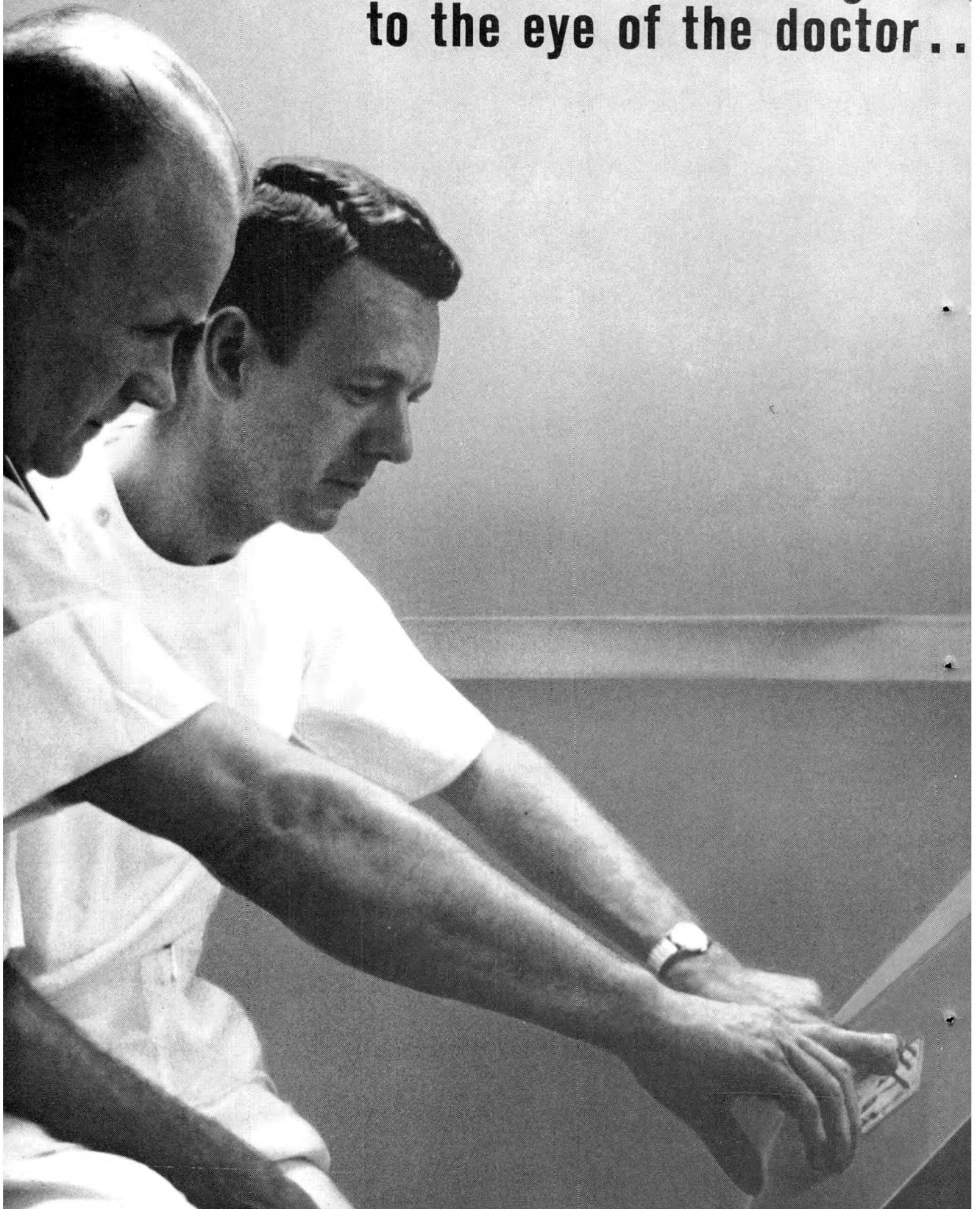


# DUNLEE

# CORPORATION



**From the hand of the technologist  
to the eye of the doctor..**





## .. an important link

A number of problems increasingly encountered in hospitals and clinics can be solved with the NEW NORELCO PROGRAMMED RADIOGRAPHIC SYSTEM. Programmed by your technologists, this "automated" system ultimately frees the technologist from repetitious equipment adjustments to concentrate on precise patient positioning and better patient care.

Once you set the program — tube, focus, auxiliary device, correct kV and corresponding optimum tube current, phototimer sensitivity and aperture are selected quickly and automatically — at the touch of a single control. "Routine," repeated examinations can be completed with astonishing speed and accuracy, duplicating best radiographic results every time.

### A FEW MORE "AUTOMATED" FEATURES:

- System X-ray control is a small selector box. That's all.
- System radiographic stands do away with tube and film centering and FFD adjustment.
- Collimator adjusts automatically to film size.
- Norelco phototiming devices and unique "falling tube load" system permit proper radiographic density in the shortest possible exposure times.

P.S. THE RESULTS SPEAK FOR THEMSELVES.



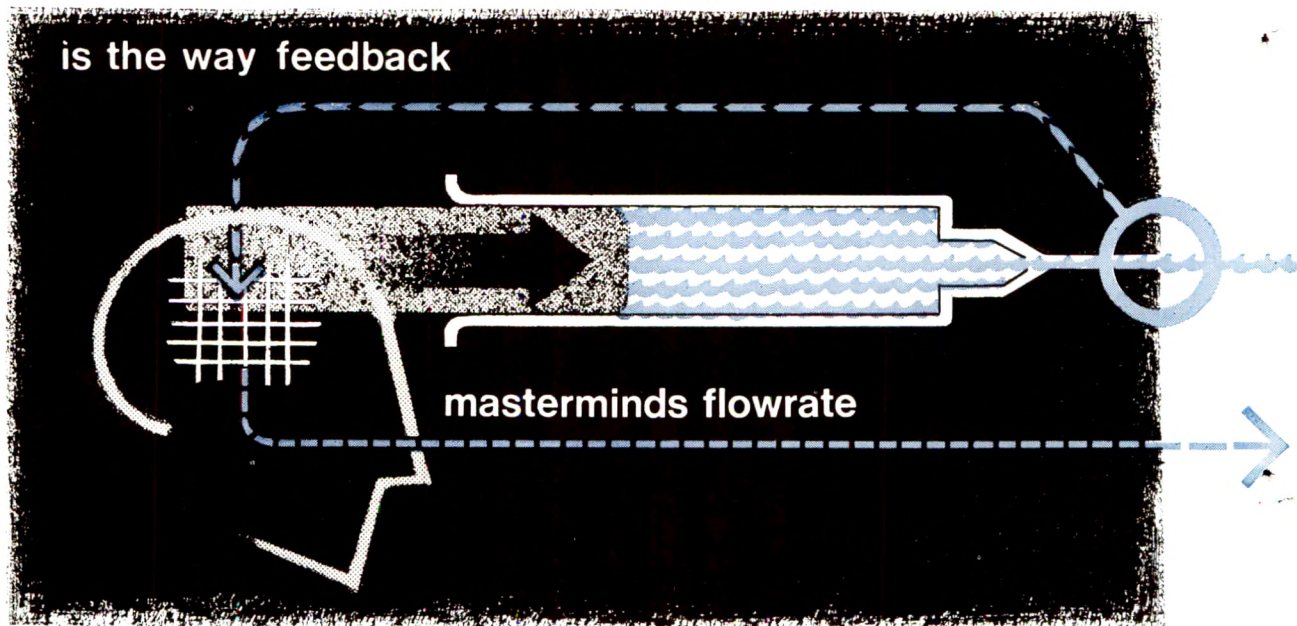
NORTH AMERICAN PHILIPS COMPANY, INC.  
Professional Products Division, 100 East 42nd St., New York, N.Y. 10017





# great virtue of the Heilman-Wholey Injector System

is the way feedback



**in angiography** the sure way to deliver the required concentration of radiopaque at the time and place you want it is to apply control *backward*, so to speak. Only by sensing what's going on at the advancing front can you take corrective measures at the rear.

*That's exactly what the Heilman-Wholey injector system does.*

A monitor circuit stands steady guard during the injection, continuously feeding back flowrate information to the syringe. Should flow slacken or speed up, it instantly signals pressure increase or decrease to maintain the flowrate selected (which can be anything from 0 to 40 cc per second). You set that rate on a single control dial. No calculations, no "resistance factors" to take into account, no hedging. *The rate you set is the rate you get.*

*Now what about safety?* Governing the flowrate is, of course, inherently safer than relying on injection pressure because it is more accurate: when resistance values must

be assumed, errors may occur. In the unlikely event of a rate failure, with consequent rapid rise of flowrate above the preset value, a safety circuit automatically stops the injection. More, the syringe employed is made of translucent polypropylene and always in full view, so you can promptly spot and deal with air bubbles, if any. And it's disposable to boot, thus precluding cross-infection risk.

**many other virtues** There's much more to say about the Heilman-Wholey injector. Like its ability to continuously control injection time duration from 0 to 6 seconds. Like the continuously variable up-to-two second x-ray trigger delay. Like the freedom to choose either a compact hand-held power injector for selective work, or an all-purpose larger one (both timed).

But that's the kind of detail more properly left to your Picker representative to tell you about. You owe it to yourself to lend him ear.

Or write Picker X-Ray Corporation, White Plains, New York.

## uniquely compact

The Heilman-Wholey control unit measures only 19" W x 11" D x 5" H. Occupying so small a space, it "fits in" wherever convenient.





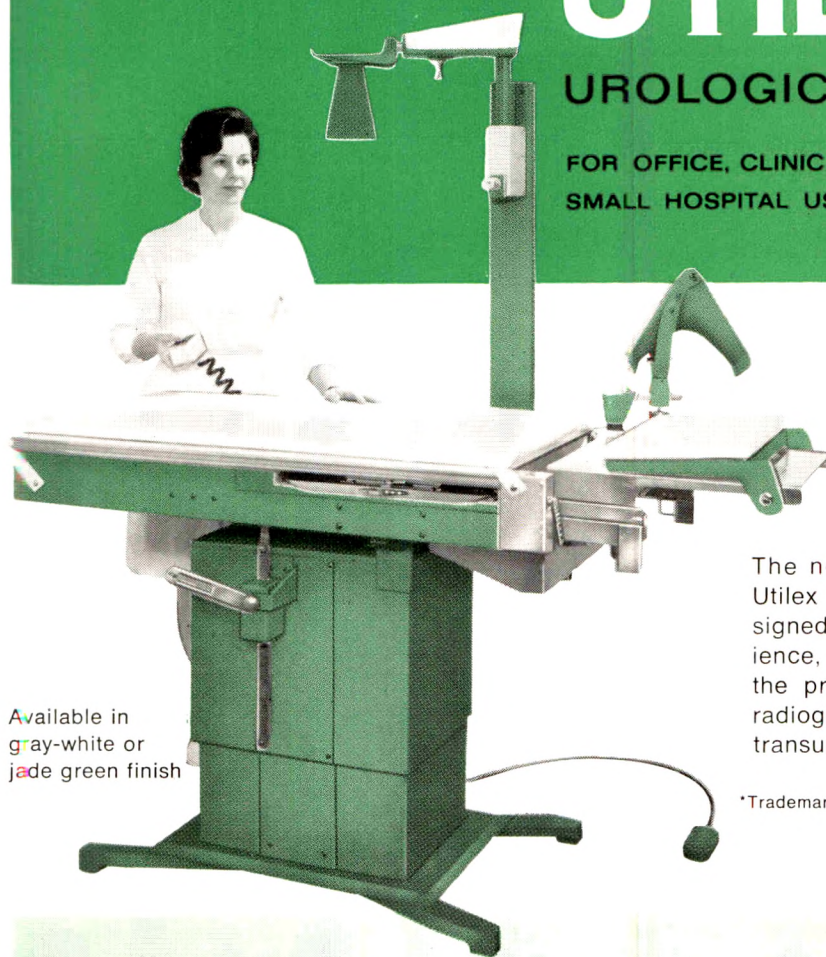
**NOW** ...in addition to the famous **HYDRADJUST**<sup>®</sup> Table

**LIEBEL-FLARSHEIM** announces the

**UTILEX**<sup>\*</sup>

**UROLOGICAL TABLE**

FOR OFFICE, CLINIC AND  
SMALL HOSPITAL USE...



Available in  
gray-white or  
jade green finish

The new moderately-priced Utilex Urological Table is designed to bring new convenience, flexibility and utility to the procedures of urological radiography, cystoscopy and transurethral surgery.

<sup>\*</sup>Trademark of Liebel-Flarsheim Company

**1967 SHOWINGS**—National and Sectional Urological Conventions and Radiological and Roentgen Ray Conventions.

For descriptive brochure and further information, contact your x-ray equipment supplier or write to—



**LIEBEL-FLARSHEIM COMPANY**  
**CINCINNATI, OHIO 45215**

A DIVISION OF RITTER PFAUDLER CORPORATION



**"...[pulmonary embolism] may exist in a grave form for a considerable time without objective signs being present on physical examination or routine chest films."<sup>1</sup>**

**P**ulmonary embolism is a mimic. Because its symptoms resemble those of other cardiorespiratory diseases—particularly myocardial infarction<sup>2,3</sup> and pneumonia<sup>4</sup>—investigators have long sought simple and certain methods of diagnosing it.

#### Many diagnostic clues—but often no clinical picture

Until a few years ago diagnosis depended primarily on the clinical history, physical findings, chest films, electrocardiograms, angiography, and pulmonary function studies. Each of these was helpful. Sometimes not even *all* of them were conclusive.

Surgery, prolonged immobilization, metastatic carcinoma and trauma often precede pulmonary embolism—but are not necessarily followed by it. Pain, dyspnea, hemoptysis may signal pulmonary embolism—but they aren't necessarily peculiar to it. The electrocardiogram may be normal in spite of it.<sup>5</sup> And there is no pathognomonic radiographic picture of pulmonary embolism.<sup>5</sup>

To be clinically valuable, however, a new diagnostic test should meet two criteria:

- it should be correlated with known pathology; i.e., it should be carefully compared with other diagnostic procedures;
- it should offer information not attainable as easily or as safely by accepted tests.<sup>6</sup>

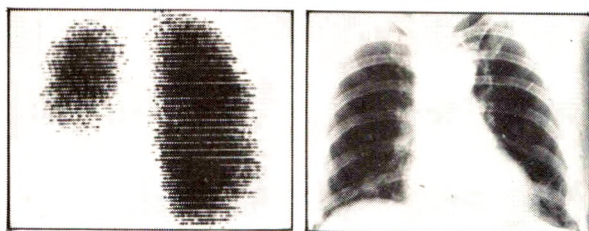
Pulmonary arteriography meets the first criterion and is a most reliable diagnostic tool. It is, however, a time-consuming procedure and one that requires experienced personnel.<sup>2</sup>

...lung scintiscanning can detect an obstruction of the pulmonary circulation as soon as it is established."<sup>7</sup>

Not only is the diagnostic procedure of lung scanning both safe and easy,<sup>2</sup> but there is information that is not attainable on chest films. "...it appears that the lung scan can point to the site of the embolic lesions before signs of lung infarction are recognizable on plain chest films."<sup>1</sup>

The scan and the x-ray shown confirm this statement.\* The photoscan of this female patient, aged 58, was taken August 13, 1965 with Albumotope-LS (Squibb Aggregated Radio-iodinated [<sup>131</sup>I] Albumin [Human]). Pulmonary emboli are clearly evident. The x-ray, taken the same day, shows no radiographic evidence of pulmonary emboli.

Lung scanning meets both criteria for a clinically valuable diagnostic test. Findings are correlated with pulmonary function studies, angiography, pathology and the clinical state of the patient. And, scanning with Albumotope-LS has been proven to be "simple, rapid, and safe in the diagnosis, localization and



ultimate fate of pulmonary emboli."<sup>2</sup> But the lung scan should not be relied upon as the only diagnostic procedure in the diagnosis of pulmonary embolism.<sup>1</sup>

**Dosage and Scanning Procedures:** Recommended scan doses of 150 to 300 microcuries of Albumotope-LS (Squibb Aggregated Radio-iodinated [<sup>131</sup>I] Albumin [Human]) depending on the instrumentation available and the technique employed. Scanning can immediately follow administration of slow intravenous injection or be delayed up to 1 to 1½ hours depending on preferred technique.

**Side Effects and Precautions:** Extensive clinical use of Albumotope-LS has not borne out the hypothetical possibility that particles of large size might induce deleterious cardiovascular or cerebrovascular effects. No antigenic properties have been specifically related to this product; one patient with a known history of angioneurotic edema, who had been given Lugol's solution in conjunction with aggregated radio-albumin similar to Albumotope-LS, developed urticaria.

Radioisotopes should not be used in pregnant women, nursing mothers, or in patients under 18 years of age unless indications are very exceptional.

**Available:** As a sterile, non-pyrogenic, aqueous suspension. Each cc. contains approximately 1 mg. aggregated human serum albumin labeled with 800-1500 microcuries of iodine-131 at time of manufacture. Also contains 0.9% benzyl alcohol as a preservative.

**References:** 1. Haynie, T. P., et al.: J. Nucl. Med. 6:613, 1965. 2. Sabiston, D.C., Jr., and Wagner, H.N.: Ann. Surg. 160:575, 1964. 3. Cooley, R.N., and Donner, M.W.: Am. J. M. Sc. 247:601, 1964. 4. Wagner, H.N., et al.: New England J. Med. 271:377, 1964. 5. Hinshaw, H.C., and Garland, L.H.: Diseases of the Chest, ed. 2, Philadelphia, W. B. Saunders Co., 1963, pp. 438-9. 6. Dworkin, H.J., et al.: Michigan Med. 64:829, 1965. 7. Quinn, J.L., et al.: Radiology 82:315, 1964. 8. Taplin, G.V., et al.: Health Physics 10:1219, 1964.

## ALBUMOTOPE®-LS

SQUIBB AGGREGATED  
RADIO-IODINATED (<sup>131</sup>I) ALBUMIN (HUMAN)

to see evidence of pulmonary embolism  
before it appears on chest films

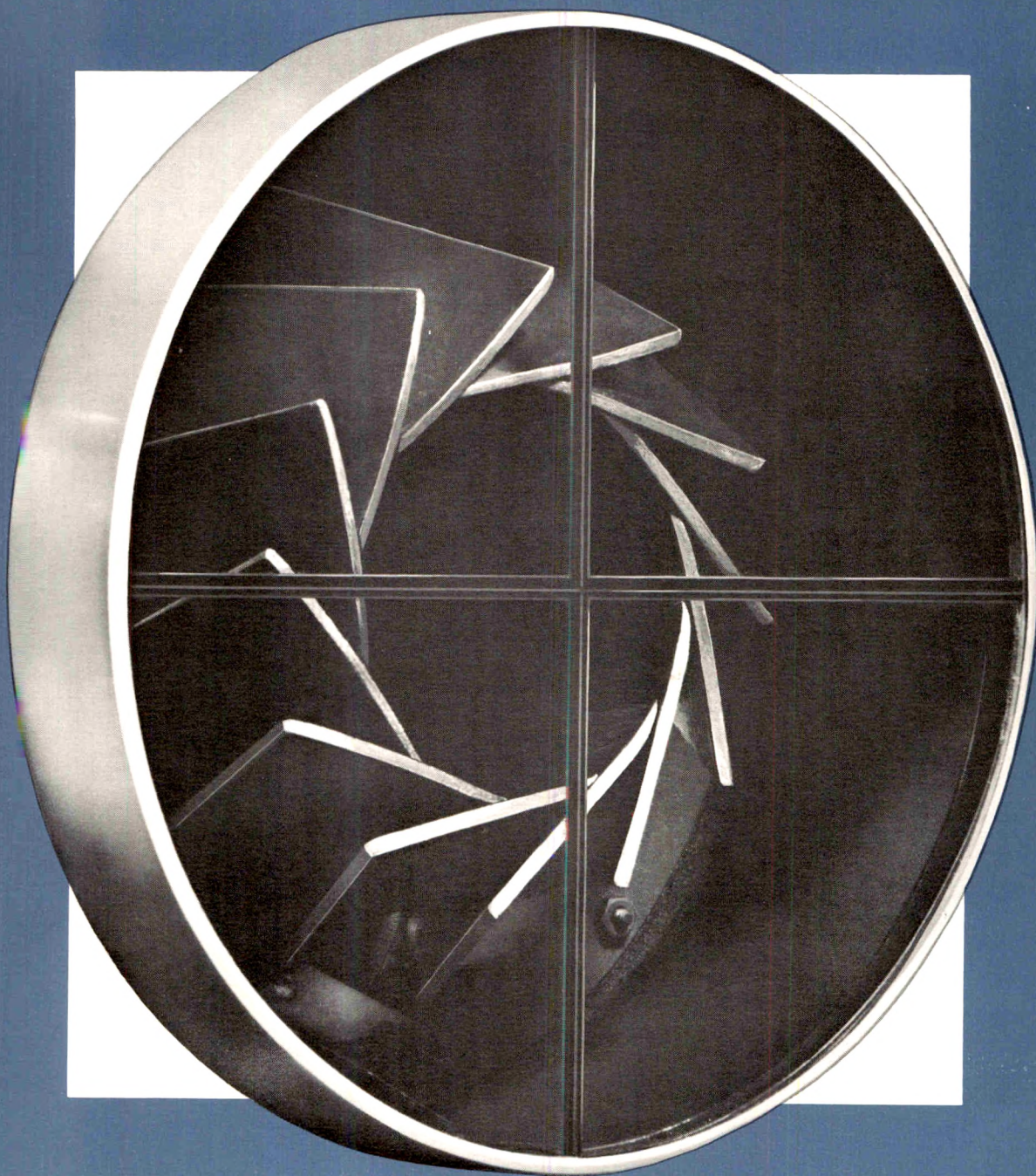
Illustrations furnished through the courtesy of George V. Taplin, M.D., Harbor General Hospital, Torrance, California.

SQUIBB



"The Priceless Ingredient" of every product is the honor and integrity of its maker





A new capability...

## **Round/Rectangular Collimation**

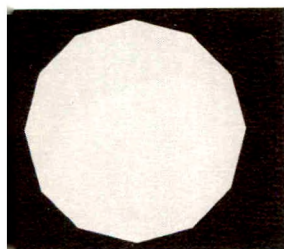
with the **Machlett-Videx Collimaster**  
to achieve a greater reduction of off-focus  
radiation combined with unusual flexibility

**MACH**

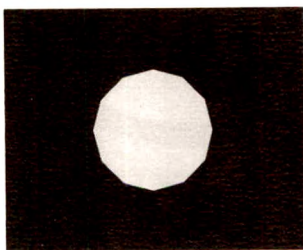




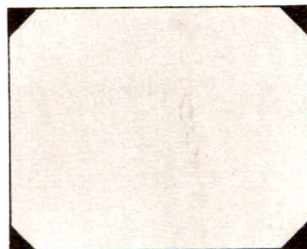
# This is Machlett-Videx Round/Rectangular Collimation: Any Geometry Without Attachments



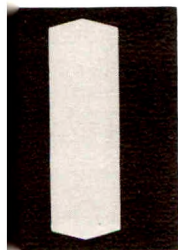
Routine  
image areas  
(all bladder)



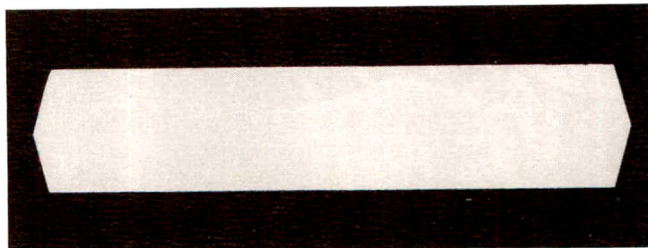
High definition  
image areas  
(sinus & mastoids)



Typical large  
area coverage  
(abdomen)



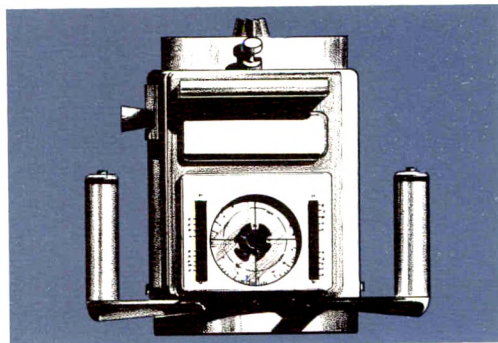
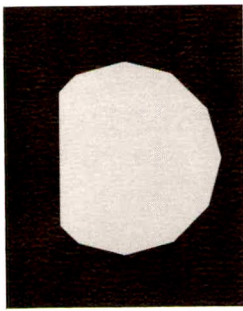
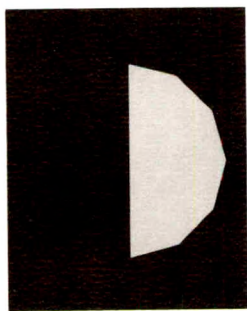
Selective  
area coverage  
(esophagus)



Special  
technique coverage  
(femoral arteriogram—14"x36" at 64")



Special beam shapes  
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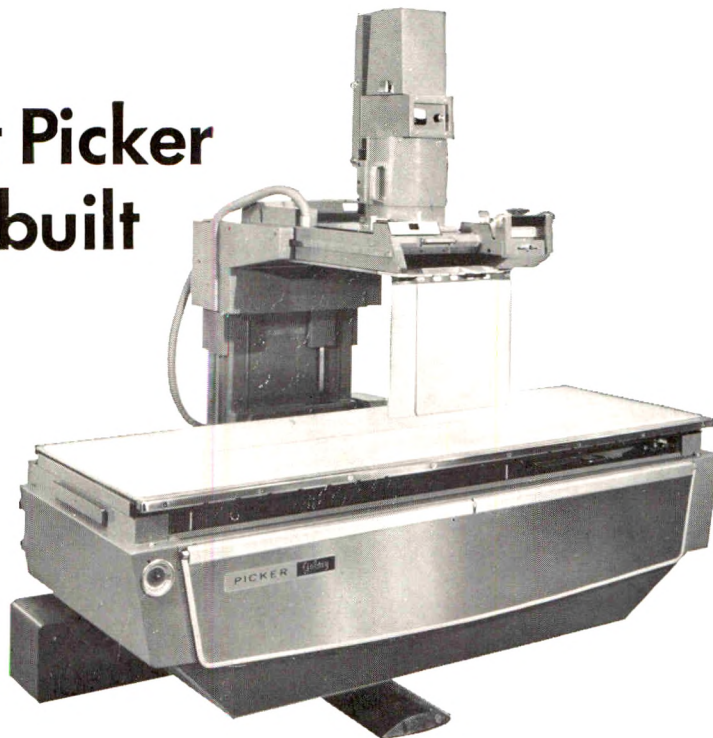
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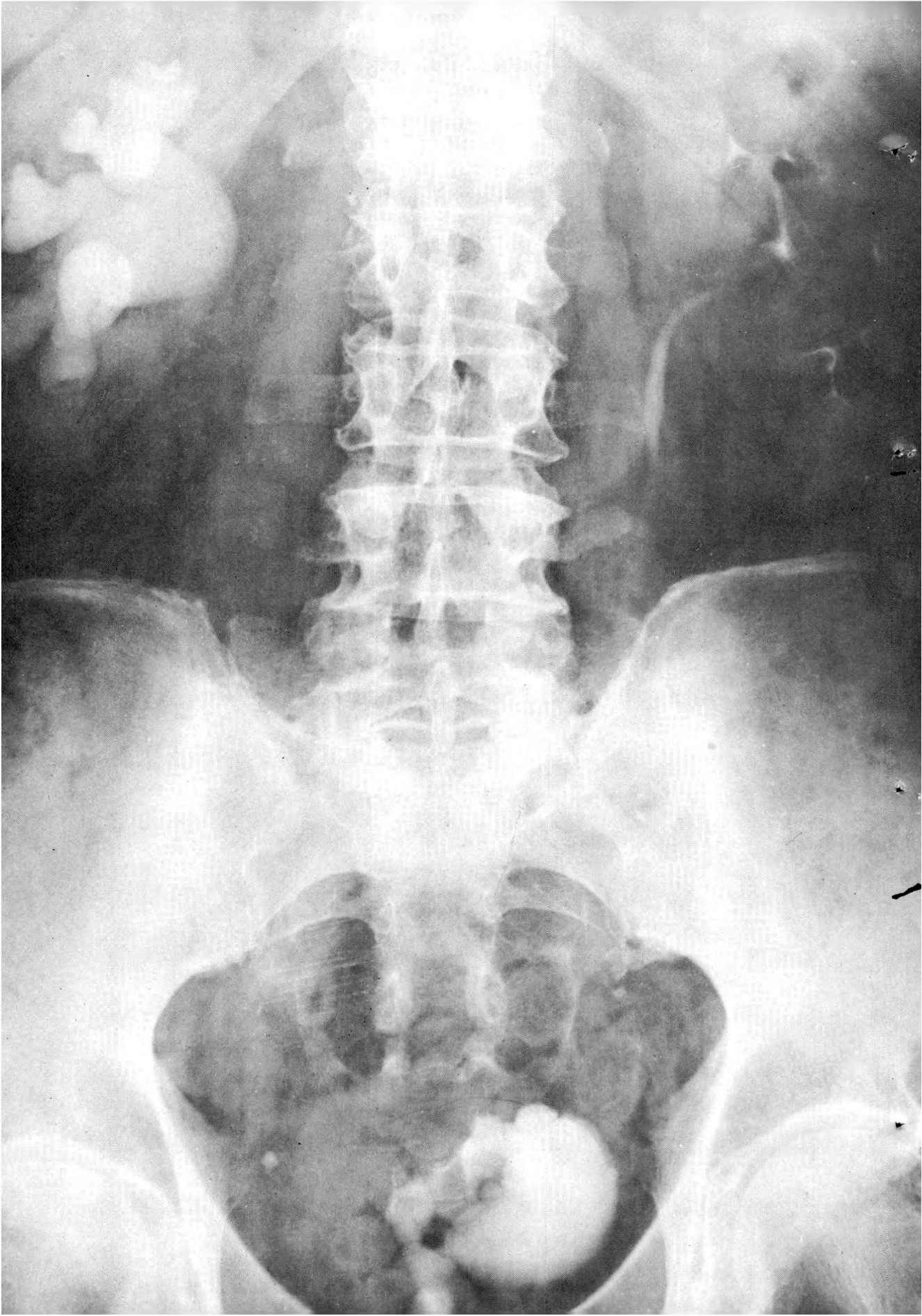
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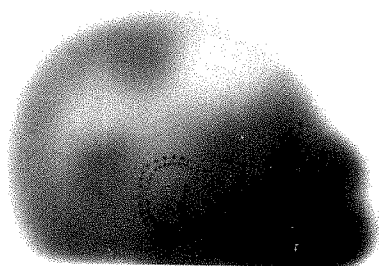
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Scan furnished through the courtesy of Lester M. Levy, M.D., Long Island Jewish Hospital, N.Y., N.Y.

Physicians who use isotopic scanning procedures look for the most reliable and informative diagnostic pictures with the least radiation danger to the patient and to themselves. Accordingly, ultrahigh energy and long-lived isotopes have tended to be replaced by lower energy and shorter-lived sources.<sup>1</sup>

### The development of isotopes with shorter, but more useful, lives

Among the isotopes of moderate energy that have acquired increased use is Technetium-99m. Having a half-life of only 6 hours, this relatively new scanning agent is obtained as the pertechnetate ion ( $TcO_4^-$ ) from Molybdenum-99, which in turn has a half-life of 67 hours.

In the two years since Harper and co-workers at Argonne Cancer Research Hospital stated that "with a clean 140-kev gamma, 6-hr.  $Tc^{99m}$  offers overwhelming physical advantages in scanning...,"<sup>2</sup> many isotope clinicians have explored its uses in discovering and localizing tumors and other abnormalities in the brain.

$Tc^{99m}$  permits rapid brain scanning and provides an image with a desirable degree of delineation. The half-life of  $Tc^{99m}$  is sufficiently short to permit intravenous doses of up to 10 millicuries without subjecting the patient to excessive radiation. It enters the blood, spreads quickly to the extracellular space, and localizes in the organ to be visualized. Selective localization of the pertechnetate ion occurs in the abnormal brain tissue sufficiently for scanning purposes.

### The problems involved in preparing $Tc^{99m}$ for use

While the basic principle of obtaining  $Tc^{99m}$  (elution from  $Mo^{99}$ ) is extremely simple, the detailed steps in assuring its sterility and freedom from pyrogens are laborious and time-consuming.

The eluent can be contaminated in a number of ways. Pyrogens may be present in the materials used in assembling a generator, in chemical reagents, in water, and other sources. They may even pass through a millipore filtration system. Nondisposable syringes may introduce contamination. Maintaining asepsis in open generator systems during elution and collection is difficult.

Even the commercial units available to laboratories have been unsatisfactory because material to be used intravenously had to be sterilized by autoclaving or filtration before injection. Pyrogens still represented an unsolved problem, since sterilization by filtration is no assurance of freedom from pyrogens.

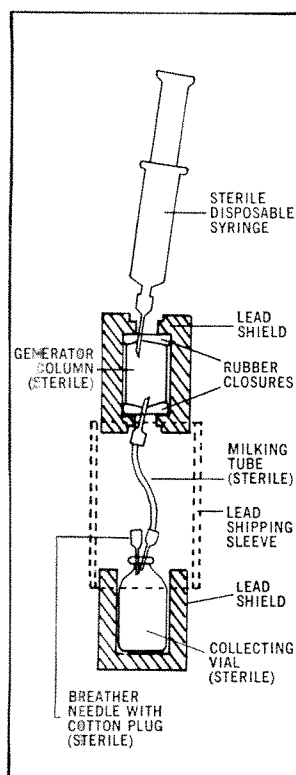
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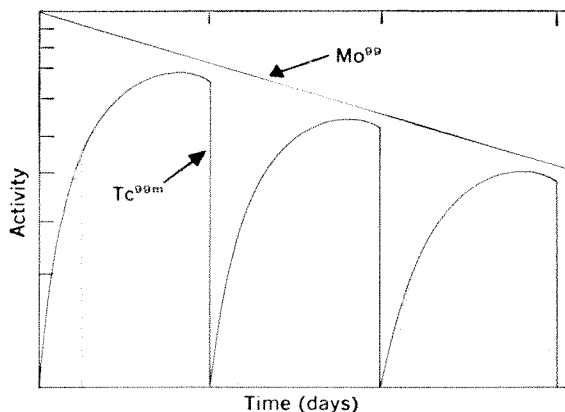
### Simplicity for the technician

The milking procedure is simple. A breather needle and one end of the double-needle milking tube are inserted into the top of the shielded collecting vial. The needle at the other end of the milking tube is inserted aseptically into the rubber closure at the bottom of the generator column. The eluent

is injected aseptically through the rubber closure at the top of the generator column. Eluted sterile  $\text{Tc}^{99\text{m}}$  will flow rapidly into the collecting vial.

The radioactivity of the  $\text{Mo}^{99}$  in the generator decays with a half-life of 67 hours. Hence the amount of  $\text{Tc}^{99\text{m}}$  in the eluate will depend upon the time interval between elutions. For optimal amounts of  $\text{Tc}^{99\text{m}}$  the generator should be eluted every 24 hours.

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Physicians and institutions with AEC licenses can obtain sterile pyrogen-free  $\text{Tc}^{99\text{m}}$  by ordering Technetope Sterile Generator, list number 08871. Write for more details to: Professional Service Department, E. R. Squibb & Sons, Inc., 745 Fifth Avenue, New York, N. Y. 10022.

**Contraindications:** Radiopharmaceuticals should not be administered to pregnant women or patients under the age of 18 unless the indications are very exceptional.

**Warning:** Maintain proper radiation safety precautions at all times.

**References:** 1. Loken, M. K., et al.: Minnesota Med. 49:237, 1966. 2. Harper, P. V., et al.: Nucleonics 22:50, 1964.

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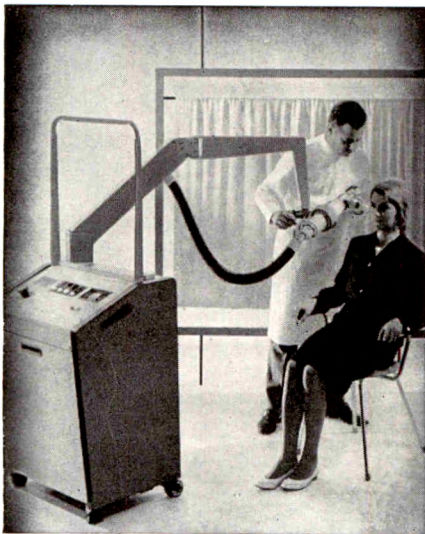
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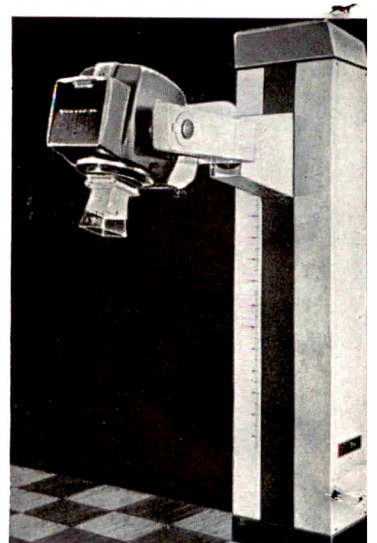
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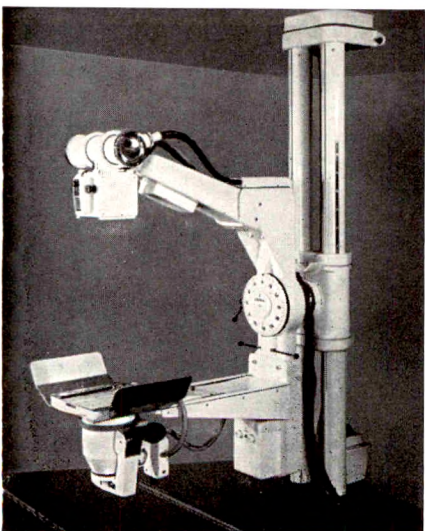
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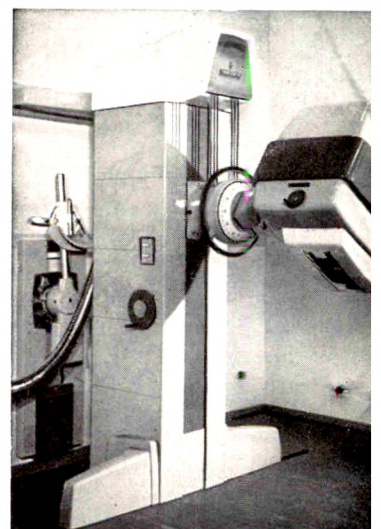


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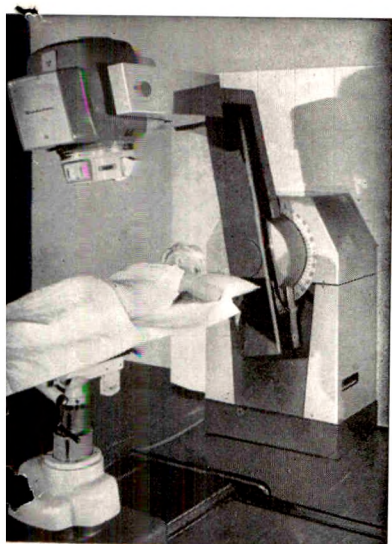
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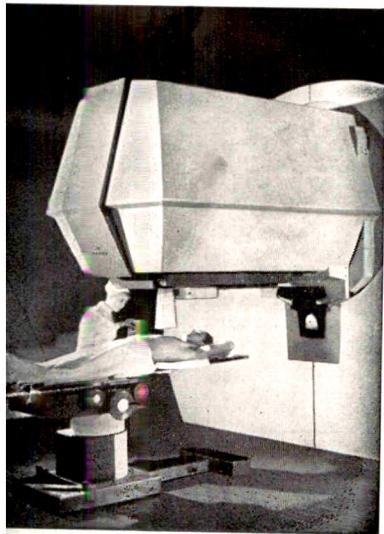


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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 98

DECEMBER, 1966

No. 4

## PROBLEMS IN RADIOLOGY OF TODAY AND THE FUTURE

### THE PRESIDENT'S ADDRESS

By CLYDE A. STEVENSON, M.D.  
SPOKANE, WASHINGTON

TODAY, the specialty of Radiology is faced with numerous serious problems, some of which threaten its very existence. Tomorrow, Radiology could be fragmented, disbursed to the winds and lose its identity. We as Radiologists know that this would be most undesirable for optimum health care of our people and it is now necessary that we take stock, analyze our strengths and weaknesses and prepare for the future.

The severe difficulties associated with federal Medicare and federal-state-Medicaid, fear of government antitrust suits and problems with physician-hospital working arrangements can, and most likely will, alter the course of Radiology. The future is most difficult to predict but we can be sure of change. Rapidly expanding scientific knowledge and wanted or unwanted added responsibilities present a host of problems which we cannot ignore if Radiology is to survive.

Our strengths are few and our weaknesses many. Our strength is due mainly to the fact that there has been a consistent increase in demand for services, so that now over one-half of the people of our

country requires the medical diagnostic or treatment skills of Radiologists. Our strength is further indicated by the full support of the American Medical Association and many specialty organizations, other than our own, in attempting to preserve our status as physicians practicing medicine in the specialty of Radiology.

A prime weakness, as amply demonstrated in today's radiological marketplace, is the present shortage of Radiologists. Supply and demand studies projected 10 years into the future indicate a progressive and severe deficiency in supply with a consistent increase in demand.

Today, in addition to increased demand for our services, our growing knowledge of special diagnostic procedures requires that we devote a relatively large amount of time to a small percent of our patients. The more common use of cobalt and high voltage roentgen therapy equipment requires that more time be spent in this aspect of our work. While these are major factors in producing our present shortage, the future is clouded by the fact that, percentage-wise, fewer physicians are now choosing



Radiology as a career. In April, 1966 the National Advisory Committee on Radiation<sup>1</sup> submitted a report to the Surgeon-General of the US Public Health Service stressing the fact that these present and future deficiencies were a serious threat to the health care of all of our people. They recommended that the Public Health Service develop and assist in several programs designed to increase the number of academic and practicing Radiologists in the United States, the number of technologists and the number of non-medical scientists associated with Radiologists in the practice of Radiology. The Committee recognized the fact that many medical school Radiology departments are understaffed and so occupied giving patient service that they have little, if any, time for research or teaching. If adequate staffing can be obtained, the Committee hopes that radiological teaching throughout the medical school years and internship would help attract more students into the specialty. It is obvious that medical school deans and faculty committees concerned with the medical curriculum must be aware of these serious present and future shortages and cooperate with their professors of Radiology in seeking solutions. I feel that, given faculty support, sufficient staff and adequate funds, either private or federal, these Radiologists are well able to develop teaching and research programs which could eventually lead to more Radiologists.

The Committee's studies showed that only about one-fourth of those selecting Radiology as a career did so before their internship. The other three-fourths were equally divided into those who selected Radiology during their internship and those who did so at a later time. Since many internships and slightly less than one-half of all positions approved for radiologic training are in hospitals not affiliated with medical schools, it is clear that non-academic Radiologists must bear

a good share of the burden of attempting to increase the number of Radiologists. Recognizing such a dual responsibility, it is incumbent on all Radiologists and their organizations to join in a cooperative effort recognizing the opportunities and needs of both the academic and non-academic divisions of our specialty. If any near-future improvement in supply can be obtained, present differences in philosophy of postgraduate training must be resolved, at least temporarily, by the Johnsonian, "Come, let us reason together."

At the present time we are blaming our failure to attract more into Radiology because we don't have time to instruct students, teach interns or maintain contact with practicing physicians, but, to potential Radiologists, the full airing of our economic and medical status problems by public news media can do little to promote interest in joining our ranks. I don't believe that these stated reasons give the entire answer but before elucidating further it is important that we recognize lack of universal agreement as to the degree of predicted shortage. Most à propos is a recent editorial, "The Per-Complex in Medicine,"<sup>2</sup> which questions the advisability of using averages of the past to project future needs for more physicians. Are the needs real, do they allow for change, and do they allow for better utilization of present personnel, space, equipment and methods?

In spite of our best efforts to attract more physicians into Radiology, analytic studies of the problem readily show that the present and estimated future needs cannot be met, and that other ways must be found for proportionately fewer Radiologists to carry the load.

What are the needs in Radiology today? No matter how many physicians we have, the increase in demand for radiological services calls for more and better technicians. We must accept most of the blame for the present very real shortage. We

<sup>1</sup> National Advisory Committee on Radiation; Report to the Surgeon General, April, 1966. U. S. Government Printing Office, 1966-o 218-937.

<sup>2</sup> Editorial, The Per-Complex in Medicine. *Pharos of Alpha Omega Alpha*, April, 1966, 29, 55.

don't want our technicians to be thoughtless "button pushers," we expect them to think as we do but we have not made their profession attractive enough to fill our needs. This problem is now well recognized and is being studied on a national basis but progress must begin at home and each employer, be he radiologist, clinic or hospital, must take the necessary steps to make x-ray technology a desirable and rewarding profession. Certainly Radiologists and patients would benefit if we follow the lead of Pathologists and establish schools leading to a bachelors degree in Radiologic Technology. It is possible that more immediate benefit would accrue from a properly financed crash program taking selected established technicians for training as chief or supervisory technicians.

A critical observer looking at the forest rather than the trees may readily see that a major need of all Radiology departments is for some major breakthrough, whereby we could consistently record roentgen images of good crisp detail without evidence of motion. Patient delay, increased cost and erroneous interpretation are commonly associated with roentgenograms showing evidence of motion. Higher kilovoltage and some overall improvement in roentgenographic equipment with improvement in films and intensifying screens represent modest progress but are far short of our ideal goal. Apparently the kilovoltage race is over in therapy equipment and special procedure hardware is becoming quite sophisticated, but for the ultimate benefit of our people we should give all encouragement and support for a major research program hopefully leading to roentgen images recorded over a period of 1/100th of a second or less in all patients. We must not be complacent about our best equipment requiring a multi-second exposure in a heavy patient.

A major advance, potentially available to us right now, depends on the acceptance by all physicians of the principle that all previous roentgenograms related to the current examination be on hand and re-

viewed before proceeding. We are quite proud of publishing a few cases of disease "X" in which an astute roentgen diagnosis led to cure of the patient, but all of us could flood the literature with instances in which lack of previous roentgenograms for review and comparison led to delay, increased cost, erroneous diagnosis, harm and even death of the patient. An intense educational program is far overdue.

Closely related to the problem of obtaining previous roentgenograms is the very common lack of clinical information giving a reason for the proposed examination. This subject has been "harped" on for many years with most Radiologists stressing its importance but a few have belittled this need. Theoretical fears that clinical information will prejudice the radiological diagnosis are greatly overshadowed by benefits which can come to the patient if the physician-radiologist knows why he is going to examine the patient and what he is looking for. Certainly the radiologist has special knowledge enabling him to determine whether or not the requested examination is the best one or if it should be modified, whether it is contraindicated or whether some other examination should precede or replace it. This knowledge is only of value if clinical information is supplied or can be obtained.

Today we still find that many Radiologists have difficulty in obtaining appropriate clinical information. Most efforts to remedy this situation have been on an individual basis and it may be that group effort on a local, state or national level would be more effective. Since the benefits to the patient and physician are so obvious and numerous, a successful program would overshadow most any of the other proposals designed to improve the radiological health care of the public. What we have failed to do over the past 50 years the federal government has done in just a few months. In the spring of this year the Federal Register elaborated on the Medicare law and stated that, in participating



hospitals, all requests for x-ray examinations of Medicare patients must contain a concise statement of reason for the examination. I hope that we don't have to wait for a national data retrieving system, closed circuit television or further federal legislation before we can get good clinical correlation and cooperation.

In spite of increase in isolation in his department, today's Radiologist working in group practice or in a closed staff hospital is usually able to maintain good communication with his relatively small number of referring physicians. They appreciate the need for various departmental routines, priorities, methods and scheduling, while in return the Radiologist communicates his findings by means of reports which usually are short, concise and to the point. However, a large per cent of our people obtain their radiological services in private offices and hospitals, where the Radiologist reports to a relatively large and heterogeneous group of doctors. Many accept roentgenological reports at their face value and place unwarranted confidence in the negative or positive aspects. In these circumstances the Radiologist needs clinical indications in order to assure maximum benefit to the patient and especially must be careful that the full significance of his report is understood by the referring physician. Today there is real need for improvement in this form of communication. It is often our only contact with practicing physicians and our image can rise or fall when the report is compared to those made by endoscopists, pathologists and others. For the past 5 years I have done a random study of typical reports from several types of radiological practices. Poor sentence construction, lack of clarity, excessive description and failure to draw conclusions were quite common. None made use of a routine overall or specific accuracy form. I feel certain that the full meaning of any report would be better understood if 3 letters and 1 numeral were placed at the conclusion. These letters are EOA for Estimated Overall Accuracy

and the numeral (1-10) to indicate the degree; *i.e.*, EOA-7 would indicate that there was a 70 per cent chance of the report being accurate. The implications and ramifications are many but this simple addition to every report could only be of benefit to all concerned and we don't have to wait for computers to do this for us. "The Roentgenologic Report" would be a good title for our scientific programs and refresher courses.

Today we are worried about our status as practicing physicians and concerned about our image but as we retreat into a maze of complicated electronic equipment we add an aura of mysticism by predicting the presence of disease if we see certain animal, vegetable or mineral "signs" on a roentgenogram. In spite of our best efforts, we continue to find "ground glass" in several parts of the body. The mystery of the roentgenogram is further heightened by our approval (or lack of objection) of poor quality, inappropriate or badly reproduced roentgenograms in the medical literature. We can be proud of the roentgenologic illustrations in our two leading journals and in a few books but our professional image suffers if occult powers seem necessary to learn anything from most of the reproductions. Some medical publications have sections devoted to a roentgenologic quiz and I am embarrassed to find that I flunk more often than pass. The quality of the reproductions is usually so poor that simple amateurish line drawings would be much better. As a specialty, we should have more than average knowledge of photography and our image is not brightened by so many ill-chosen and badly reproduced roentgenograms. Irregardless of the origin of the literature, it is imperative that we offer our services to editors and publishers to review roentgenographic illustrations before publication.

The knowledge explosion in Radiology, while highly beneficial in the health care of the public, has resulted in increased subspecialty activity with more Radiologists limiting their work to a relatively

small segment of Radiology. Today, the general Radiologist is faced with the almost impossible task of being current in his knowledge and practice. A good share of the public is served by solo or small group Radiologists who find it impossible to be expert in all branches. In view of the dim outlook for any marked increase in radiological manpower I can see no easy solution or corrective action. In discussing the future of Radiology, Millis<sup>2</sup> has said that in order to be a competent "generalist" one probably has to be brighter, better educated, more energetic and more experienced than the specialist. In order to meet the needs of the public, we must continue to train radiological generalists and perhaps Program Directors can be guided by Dr. Millis' suggestions, although a solution or even a compromise is not evident, for we also want our best residents to be the teachers of the future.

In conclusion, it is easy to note that I have presented very little new. The need for more technicians and better equipment

is obvious. No physician likes to do a physical examination without knowing the patient's history and I believe that we have abrogated some of our physician prerogatives by not insisting on our special type of history; namely, previous roentgenograms for study and adequate clinical information. We also don't act like physicians if we use bizarre and exotic terminology to report our findings and our reports don't always say just what we mean. Some of these deficiencies are bound to alter our image of being practicing physicians and thus make a career in Radiology less attractive to students, interns, and others. Any person reading the medical literature must be confused by most of the roentgenographic illustrations for he cannot see what he apparently is supposed to see. Can we blame him if he gets a somewhat distorted image of Radiology?

I have touched on only a few of the problems we are faced with today; the challenges of the future will be difficult to meet.

Department of Radiology  
Sacred Heart Hospital  
Spokane, Washington 99204

<sup>2</sup> MILLIS, J. S. The Future of Radiology. *Proc. Am. Coll. Radiol.*, Feb. 5, 1966.





## SUPRAVALVAR AORTIC STENOSIS\*

### ROENTGEN ANALYSIS OF TWENTY-SEVEN CASES

By GERALD J. KURLANDER, M.D., EUGENE L. PETRY, M.D., HOOSHANG TAYBI, M.D.,†  
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**S**TENOSIS of the lumen of the ascending aorta distal to the origin of the coronary arteries has been recognized with increasing frequency in recent years. Although Chevers<sup>7</sup> was aware of the condition in 1842, the earliest known angiographic diagnosis was made in 1953 by Campbell and Lurie (Fig. 1).

Thirty-eight cases of congenital supra-valvar aortic stenosis (SVAS) have been studied at the Indiana University Medical Center since 1960.\* The purpose of the authors is to describe the roentgen experience with 27 patients who had both cardiac catheterization and cinecardioangiography. Clinical, hemodynamic, and genealogic features of the larger group will be presented in greater detail elsewhere. As a background for this presentation, a review is made of the pathology and the conditions associated with stenosis of the ascending aorta.

#### PATHOLOGY

Several cases of nonobstructing bands or cords located at the level of insertion of the aortic valve cusps have been reported.<sup>2,22,24</sup> Most of these bands traverse the aortic lumen between the commissure of the left and noncoronary cusps, and that of the right and left cusps. There is no known example of significant aortic obstruction from such a band.

Thin supra-valvar membranes or fibrous diaphragms located at the upper margins of the sinuses of Valsalva are rare causes of obstruction.<sup>6,15,19</sup> The openings in these

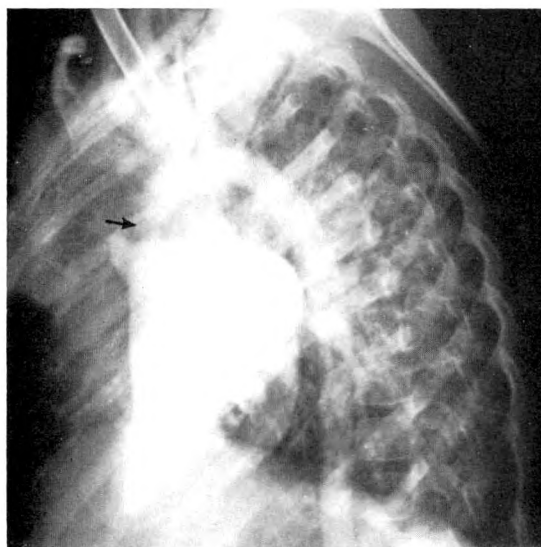


FIG. 1. M.L. I.11-2. Five year old retarded male. Intravenous angiogram in the left anterior oblique position demonstrating severe supra-valvar aortic stenosis (arrow). September 21, 1953.

membranes or diaphragms are said to be central or eccentric. Distal to the level of obstruction, the aorta may be of normal caliber or may be dilated. The membranous or diaphragmatic type of obstruction of the ascending aorta has not been encountered at this institution.

Almost all cases of SVAS have had greater or lesser degrees of thickening of the plica at the upper margins of the sinuses of Valsalva. The thickened plica protrudes into the lumen to effect narrowing. Few histologic studies of the aortae in cases of SVAS have been reported. The aorta at the level of maximal narrowing reveals both fibrous intimal proliferation and medial hypertrophy. The ascending aorta, when

\* This study does not include isolated stenosis of the aortic lumen distal to the ascending aorta or uniform narrowing or hypoplasia of the entire aorta.<sup>1</sup>

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involved distal to this area, shows predominantly medial hypertrophy. The thick media has an abnormal arrangement of fibers in a mosaic pattern.<sup>9</sup>

In a recent review of the literature, Peterson *et al.*<sup>20</sup> found that of 43 cases of SVAS, in which the aortic valve was described anatomically, 30 had normal aortic valves. Of the remaining 13 cases, 8 showed evidence of adhesion of part or all of the free edge of one or more cusps to the supravalvar ridge. Of 51 cases in which the condition of the aortic arch was mentioned, 8 had stenosis of a brachiocephalic trunk.

Since the coronary arteries are proximal to the obstruction in SVAS, they are in a high pressure zone. During systole the coronary arteries are subjected to high intraluminal pressure but flow is impeded by the contraction of hypertrophied myocardium. This combination of forces produces distention of coronary arteries which may result in anatomic changes. Of 33 cases reviewed by Peterson *et al.* in which the anatomic condition of the coronary arteries was mentioned, 20 had abnormalities. These abnormalities included: (1) narrowing at or just distal to the coronary ostium, (2) increased external diameter and tortuosity associated with medial thickening, fibrous intimal thickening and sometimes atherosclerotic intimal changes, and (3) thin walled, histologically normal, coronary arteries when protected from high intraluminal pressure by the adherence of the free edge of the associated aortic cusp to the site of SVAS.

In the group of 68 cases of SVAS reviewed by Peterson *et al.*, there were 13 instances of postvalvar pulmonary artery stenosis, among the 19 in which the status of the pulmonary arteries was mentioned.

Gross anatomic observations were made by one of us (E.L.P.) at the operating table in the 11 surgically treated cases from our group of 27 cases of SVAS. In 8, the wall of the ascending aorta distal to the upper margins of the sinuses of Valsalva was thickened. Seven of the 8 had a thick circumferential ridge producing localized nar-

rowing at the upper margins of the sinuses. Distal to this level, the aortic lumen increased (accompanied by decrease in wall thickness), reaching normal or near normal size proximal to the innominate artery in 4 cases. In the other 4 cases, however, the thick wall and associated narrowing extended into the arch and was associated with narrowing of one or more brachiocephalic trunks. The thick aortic wall and marked narrowing of the lumen distal to the supravalvar ridge contributed to the over-all obstruction to ejection from the left ventricle. In 1 of the 8 cases, there was no discrete supravalvar ridge but rather nearly uniform narrowing of the entire ascending aorta associated with thickening of the aortic wall. The aortic valve in each of these 8 cases was normal.

Three of the 11 surgically treated cases differed in several ways from the 8 referred to above. Each had a mild to moderate circumferential constriction just above the upper margins of shallow sinuses of Valsalva. The intima at the level of constriction was slightly thickened but distal to this level the aortic wall was of normal thickness. The aortic valves in these 3 cases, in contrast to those described above, were thick and markedly limited in mobility but, most significantly, the commissures were not fused. Angiographically, there were certain differences between one of these cases and the other two which will be referred to later.

#### ANGIOGRAPHIC CLASSIFICATION OF SUPRAVALVAR AORTIC STENO- SIS (Fig. 2)

Type 1a. Short ascending aorta with narrowing of the entire ascending aorta maximal at the upper margins of dilated sinuses of Valsalva.

Type 1b. Ascending aorta of normal length with narrowing of the proximal one-third maximal at the upper margins of dilated sinuses of Valsalva. Normal or dilated aorta above the narrowed area.

Type II. Mild to moderate localized narrowing just above shallow sinuses of Val-



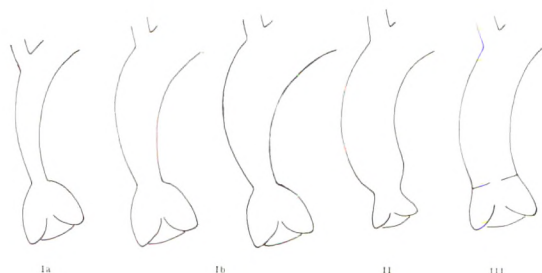


FIG. 2. Diagrammatic representation of the 3 types of supra-valvar aortic stenosis.

salva. Aortic dilatation distal to narrowing. Thick, semirigid, unfused aortic valve cusps with small annulus.

Type III. Thin membranous or fibrous diaphragm located at the upper margins of the sinuses of Valsalva. Normal or dilated ascending aorta.

#### CLASSIFICATION OF POSTVALVAR PULMONARY ARTERY STENOSIS (PPAS)

Type I. Stenosis involving the main pulmonary trunk, bifurcation, right or left main pulmonary artery.

a. Single localized stenosis.

b. Multiple localized stenoses or diffuse stenosis.

Type II. Stenosis peripheral to right or left main pulmonary artery.

a. Single peripheral stenosis.

b. Multiple localized stenoses or diffuse stenosis.

Type III. Any combination of Type I and II.

#### ROENTGEN FINDINGS

##### PLAIN ROENTGENOGRAMS

Of 24 cases of Type I SVAS, 18 had normal heart size and 6 had slight to moderate cardiac enlargement. Of the latter 6, the frontal configuration of the heart suggested left ventricular enlargement in 4, right ventricular enlargement in 1, and left ventricular and biatrial enlargement in 1 (Fig. 3, A and B). Of 3 patients with left atrial enlargement, all had large left ventricles and resting left ventricular-aortic systolic pressure gradients of more than 150 mm. Hg. Right ventricular enlargement occurred in an individual with mild

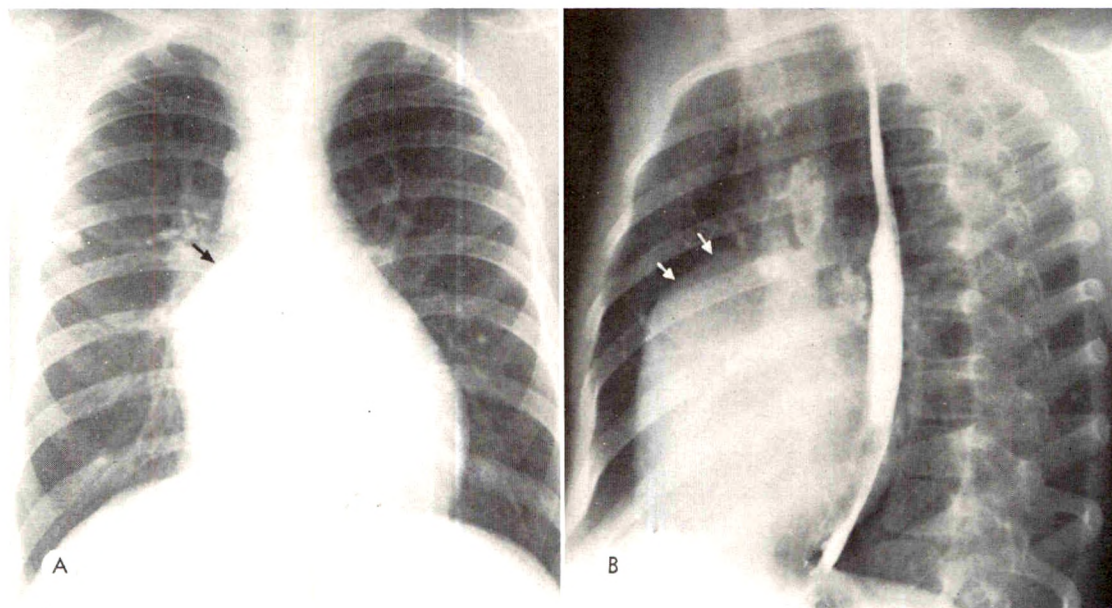


FIG. 3. R.S. M.11-2. Eleven year old retarded male with Type Ia SVAS. (A) Frontal chest roentgenogram with barium in the esophagus. Dense right paratracheal lymph node calcification is seen. The superomedial margin of the right atrium (arrow) is unusually well visualized as a result of the small medially placed ascending aorta. The aortic impression on the esophagus is slight. (B) Left anterior oblique chest roentgenogram with barium in the esophagus. Note the prominent right atrial margin (arrows).

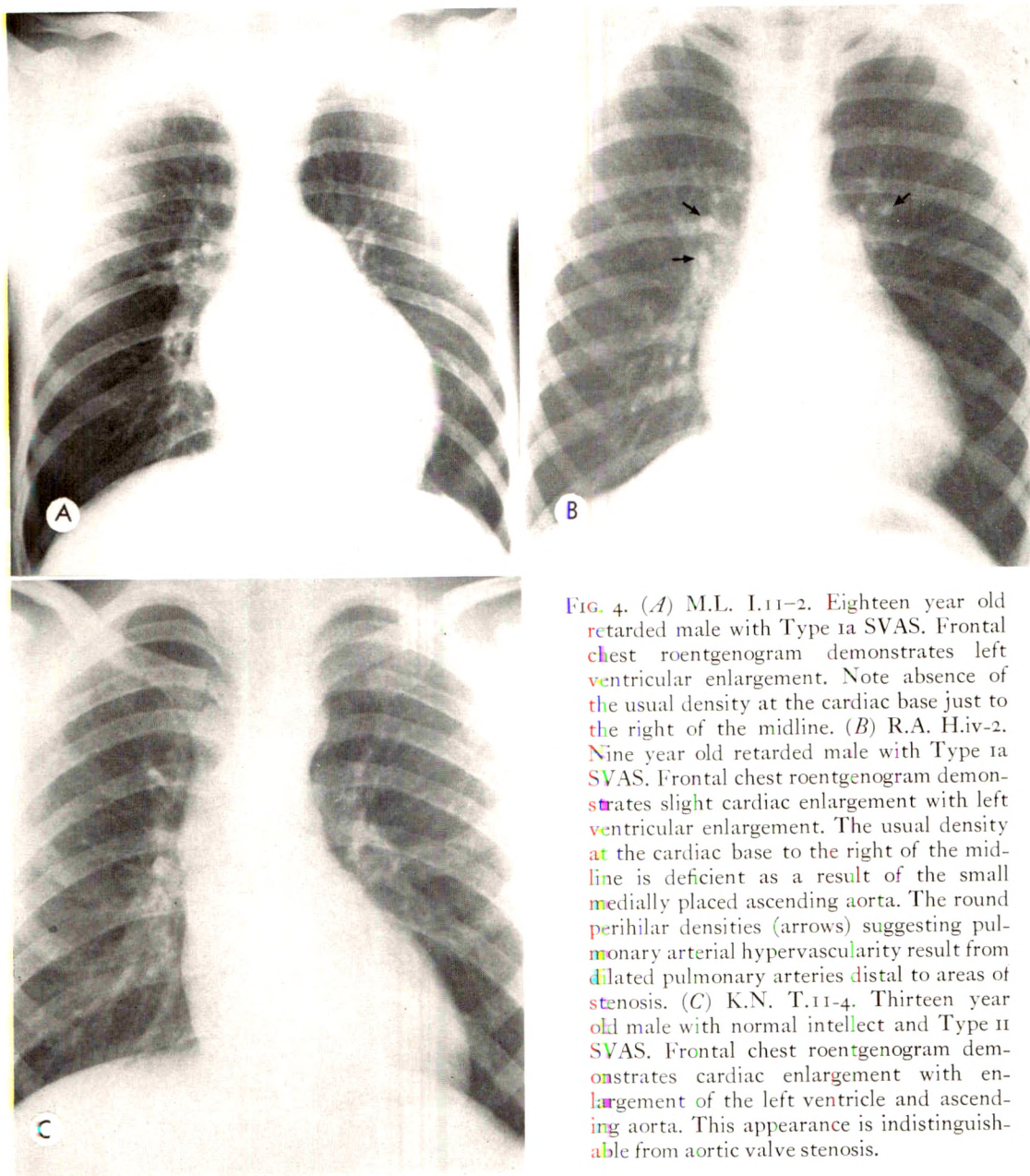


FIG. 4. (A) M.L. I.11-2. Eighteen year old retarded male with Type Ia SVAS. Frontal chest roentgenogram demonstrates left ventricular enlargement. Note absence of the usual density at the cardiac base just to the right of the midline. (B) R.A. H.iv-2. Nine year old retarded male with Type Ia SVAS. Frontal chest roentgenogram demonstrates slight cardiac enlargement with left ventricular enlargement. The usual density at the cardiac base to the right of the midline is deficient as a result of the small medially placed ascending aorta. The round perihilar densities (arrows) suggesting pulmonary arterial hypervascularity result from dilated pulmonary arteries distal to areas of stenosis. (C) K.N. T.11-4. Thirteen year old male with normal intellect and Type II SVAS. Frontal chest roentgenogram demonstrates cardiac enlargement with enlargement of the left ventricle and ascending aorta. This appearance is indistinguishable from aortic valve stenosis.

SVAS but with severe postvalvar pulmonary artery stenosis.

The best clues to the diagnosis of Type I SVAS are reduction in the usual density noted superior to the right atrium and just to the right of the midline on the frontal chest roentgenogram with fluoroscopic evidence of diminished aortic pulsation (Fig. 4A). These findings correlate well with a small, rather medially

placed ascending aorta and were present in 38 per cent (9 of 24) of cases of Type I SVAS. The aortic impression on the esophagus was small or absent in each of these 9 cases. Eight of the 9 had resting left ventricular-aortic systolic pressure gradients of more than 65 mm. Hg, indicating severe SVAS.

In 3 cases, plain roentgenograms of the chest suggested enlargement of the right



atrium but only 1 of these was associated with cardiac enlargement. There are several possible explanations for this finding. When the ascending aorta is small, more of the superior margin of the right atrium is seen, giving the false impression of right atrial enlargement. Elevated right ventricular

pressure from postvalvar pulmonary artery stenosis could also explain right atrial enlargement in some instances. Since the aortic obstruction is above the coronary arteries, they are subjected to a high perfusion pressure and tend to become dilated. We have observed dilated coronary sinuses

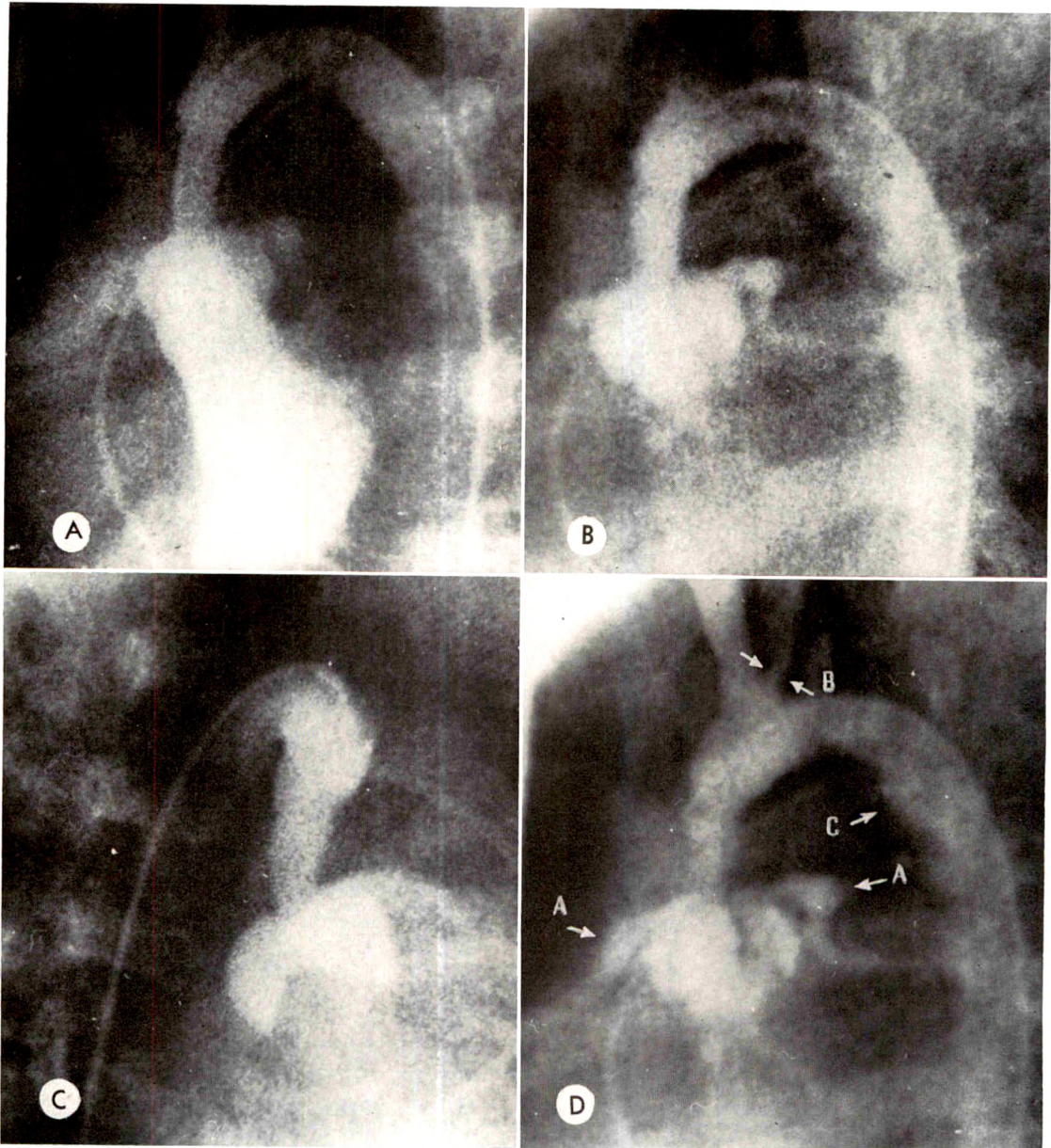


FIG. 5. L.B. F. 111-2. Fourteen year old male with normal intellect and Type Ia SVAS. Cinerentgenographic study. (A) Left ventriculogram in the left anterior oblique position and aortograms in the (B) left and (C) right anterior oblique positions demonstrate a short ascending aorta with marked narrowing maximal at the upper margins of dilated sinuses of Valsalva. (D) Aortogram in the left anterior oblique position shows dilated coronary arteries (A), narrowing of the origin of the left common carotid artery (B) and a ductus diverticulum (C).

on cinecardioangiograms in several cases of SVAS where the coronary arteries were large. Perhaps truly large right atria may, in some instances, result from the development of coronary arteriovenous fistulae after years of high perfusion pressure. This has not been observed in any of our cases, however.

Frontal chest roentgenograms of 3 cases suggested increased arterial vasculature in the lungs. One occurred in a case proven to have an interatrial septal defect. The prominent pulmonary arteries in the other 2 cases were shown to be dilated arteries beyond multiple areas of narrowing (Fig. 4B). In one instance, postvalvar pulmonary artery stenosis was suspected because of localized dilatation of several peripheral pulmonary arteries. The main pulmonary artery was not enlarged in this case but accentuated pulsation was noted fluoroscopically at the time of cardiac catheterization.

Two of the 3 cases with Type II SVAS had cardiac enlargement but all 3 had rounded plunging cardiac apices, indicating left ventricular enlargement (Fig. 4C). These 3 cases differed from those with Type I SVAS in that they had moderate to marked dilatation and accentuated pulsation of the ascending aorta.

#### CARDIOANGIOGRAPHIC FINDINGS

Cardiac catheterization and 16 mm. cinecardioangiographic studies were performed in each of the 27 cases. Left ventriculography was performed in each of the 27, thoracic aortography in both the left anterior and right anterior oblique positions in 26 and pulmonary arteriography in 25. In addition, many had pulmonary arteriograms in both the posteroanterior and oblique positions and thoracic aortograms in the posteroanterior position. The number of contrast injection film sequences per patient varied from 3 to 7 (average 5).

As indicated earlier, no case of a membranous or fibrous diaphragmatic type of obstruction of the supravalvar segment of the aorta was encountered.

Ten cases of Type Ia SVAS were studied. This group is characterized angiographi-

cally by a short ascending aorta as judged by the distance between the aortic valve and the origin of the innominate artery (Fig. 5, A-D). Narrowing is most severe at the upper margins of the sinuses of Valsalva, but the entire ascending aorta is reduced in caliber. No case of generalized narrowing of the entire aorta was encountered. The area of maximal narrowing in the ascending aorta was produced by a ridge detected at the time of surgery in 7 of the 9 operated on cases of Type Ia. One case without a supravalvar ridge had nearly uniform narrowing of the entire ascending aorta to the level of the innominate artery (Fig. 6, A and B).

Pulsations of the ascending aorta were diminished or absent in cases of Type Ia SVAS, a finding which correlates with the marked thickening of the ascending aortic wall noted at the time of surgery in the 9 operated on cases.

The angiographic appearance of the aortic valve was normal in 9 of the 10 cases of Type Ia SVAS. The aortic sinuses and both of the coronary arteries were dilated in all 10 cases. With one exception, Type Ia SVAS had the largest coronary arteries and the greatest dilatation of the sinuses of Valsalva. Five of the 10 cases of Type Ia had stenosis of a brachiocephalic trunk and 3 had dilatation of the innominate artery. Ductus diverticula reported in several instances of SVAS<sup>3,14</sup> were seen in 3 cases with Type Ia. Two cases had aortic coarctation just distal to the left subclavian artery and 1 other had absence of the left renal artery and left kidney. Postvalvar pulmonary artery stenosis was demonstrated in 5 of the 10 cases.

In 1 case, aortograms demonstrated a short ascending aorta, narrowing at the upper margins of the sinuses of Valsalva, systolic doming of the aortic valve and moderate dilatation of the sinuses. Surgical inspection showed thick, semirigid aortic cusps which had no fusion of their commissures. In our series, the only instance of ventricular septal defect occurring with SVAS was in this one patient with atypical Type Ia stenosis.



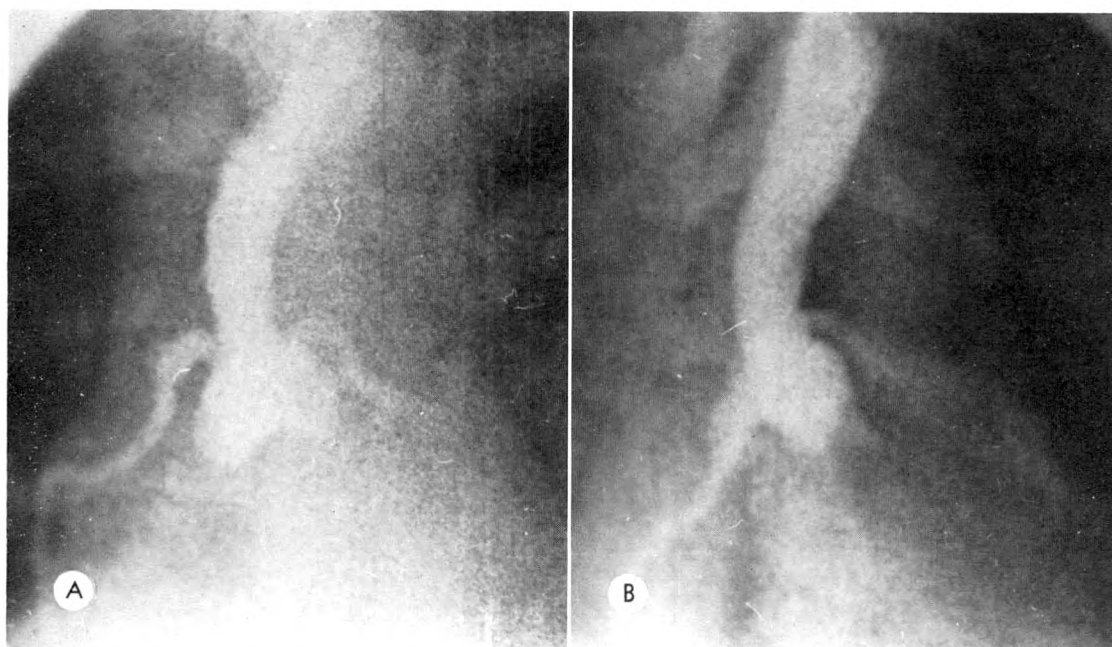


FIG. 6. D.McC. A.111-14. Eleven year old female with normal intellect and Type 1a SVAS. Cineroentgenographic study. Aortograms in the (A) mild left anterior oblique and (B) right anterior oblique positions demonstrate nearly uniform narrowing of the ascending aorta. The coronary arteries and sinuses of Valsalva are dilated.

All 10 cases had loud, systolic ejection murmurs and thrills, maximal in the supra-sternal notch or in the neck. The resting left ventricular-aortic systolic pressure gradient exceeded 60 mm. Hg in every instance and in 5 of the 10 was more than 100 mm. Hg (Table 1).

Type 1b SVAS is, in our experience, a milder form of aortic narrowing. The ascending aorta is of normal length. The area of narrowing at the upper margins of the sinuses of Valsalva is milder than in Type 1a and the vessel widens to a normal or slightly increased caliber at the level of the mid ascending aorta (Fig. 7, A, B and C; and 8, A and B). Systolic expansion is absent or minimal in the proximal ascending aorta but 10 of the 14 cases of Type 1b had increased pulsation in the mid and distal ascending aorta.

Of 14 cases with Type 1b SVAS, 11 had dilatation of the sinuses of Valsalva and 12 had enlargement of both coronary arteries. In general, sinus dilatation and coronary artery enlargement were less marked than in Type 1a.

Two cases with Type 1b had stenosis of a brachiocephalic trunk and a third had bilateral renal artery stenosis detected by abdominal cine-aortography (Fig. 9, A-F). In 2 other instances, the right aortic cusp was considered abnormal because of restriction of movement. One of these 2 cases had moderate aortic insufficiency. Mild coarctation of the aorta distal to the left subclavian artery was discovered in 2 cases and 1 other case had a ductus diverticulum. The only intracardiac anomaly found in this group was 1 case with an interatrial septal defect. Thirteen of the 14 cases of Type 1b SVAS had postvalvar pulmonary artery stenosis.

In every case with Type 1b SVAS, the resting left ventricular-aortic systolic pressure gradient was less than 70 mm. Hg. This gradient increased to more than 60 mm. Hg in 5 of 8 cases during infusion of isoproterenol.\* Systolic ejection murmurs were less intense and thrills less common than in Type 1a (Table 1).

Three examples of the lesion which we

\* An agent which increases cardiac output by decreasing peripheral resistance and increasing cardiac rate and contractility.

have chosen to call Type II SVAS have been encountered. Mild to moderate narrowing of the aorta just above shallow sinuses of Valsalva was observed in each instance (Fig. 10, A-D). Distal to the area of supravalvar narrowing, the ascending aorta was dilated and pulsated excessively. The aortic valve cusps were thick and restricted in mobility but without fusion of commissures. No case in this group was associated with postvalvar pulmonary artery stenosis or brachiocephalic trunk abnormality. In each of the 3 cases, the aortic valve annulus appeared smaller than normal. One of the 3 had severe localized aortic coarctation just distal to the left subclavian artery.

#### CONDITIONS ASSOCIATED WITH SVAS

SVAS, *mental retardation, and peculiar facies*. This syndrome has been recognized with increasing frequency since its original description in 1961.<sup>25</sup> The characteristic facial features are: depressed nasal bridge, wide set eyes, wide nasal philtrum, thick pouting upper lip, pointed chin, and small mandible (Fig. 11, A and B). Beuren *et al.*<sup>3</sup> recognized specific dental malformations in their 10 cases with abnormal facies. Typical examples of the abnormal dentition include hypoplastic crowns and short, thin roots. The broad upper oral arch overlaps the small mandible with resulting dysgnathism. Low birth weight, strabismus, and inguinal hernias are also frequently noted.

The behavior of these children is friendly although they are moderately to severely retarded. Eight of our 9 cases with this syndrome had mild microcephaly. Chromosome studies were normal in all 5 of the cases in which the examination was performed.

Of the 9 cases with this syndrome, 4 had Type Ia SVAS and 5 had Type Ib. Seven of the 9 had postvalvar pulmonary artery stenosis.

*Supravalvar aortic stenosis and idiopathic infantile hypercalcemia*. In 1952, Fanconi *et al.*<sup>11</sup> described 2 children with an unusual syndrome of dwarfism, mental retardation, strabismus, osteosclerosis, hypercalcemia,

and abnormal renal function. Subsequently, other cases of idiopathic infantile hypercalcemia have been reported. Many of these patients had cardiac murmurs, but in a review of 53 published cases Rashkind *et al.*<sup>21</sup> found none with supravalvar aortic stenosis.

Black and Bonham Carter<sup>4</sup> in 1963 suggested that the facies of cases with idiopathic infantile hypercalcemia were strikingly similar to those of patients with the syndrome of SVAS, mental retardation and abnormal facies. Since 1963, there have been well documented case reports of idiopathic infantile hypercalcemia with SVAS,<sup>13</sup> with SVAS and PPAS, and with PPAS.<sup>14</sup> Whether most or all cases with SVAS and/or PPAS, mental retardation, and peculiar facies represent individuals who have or at one time had idiopathic infantile hypercalcemia is unknown at this time.

Our cases did not have blood calcium studies in infancy, but no examples of osteosclerosis were encountered. Our series, however, includes only 2 individuals who were 3 years of age or under when cardiac catheterization and cinecardioangiography were performed. Chest roentgenograms in 2 other cases when less than 1 year of age did not show osteosclerosis. It is of interest that neither the dental anomalies seen in the retarded patients with peculiar facies and SVAS nor the familial occurrence seen in those with SVAS and normal intellect or mental retardation have been associated with idiopathic infantile hypercalcemia.

*Familial SVAS with normal intellect*. With a few notable exceptions (R.A.—H.IV-2 and C.A.—H.IV-3, in our series and Vogel and Blount's<sup>23</sup>), well documented familial SVAS occurs with normal facies and, apparently, normal intellect. In at least 4 previous reports,<sup>10,12,16,26</sup> including that of Logan describing 6 cases in 3 generations of the same family, examples of familial SVAS have been presented.

Thirteen of our cases occurred in 6 unrelated family groups. In 4 of the families, the occurrence of more than 1 individual with SVAS was documented by cardiac catheterization and cinecardioangiography.



TABLE I

TABULAR SUMMARY OF PLAIN FILM, CLINICAL, CARDIOANGIOGRAPHIC, AND CARDIAC  
CATHETERIZATION DATA OF 27 CASES OF SUPRAVALVAR AORTIC STENOSIS

		Plain Film Findings				Clinical Data										
		Heart Size	Chamber Size	Ascend- ing Aortic Size	Pulmo- nary Vascu- lature	Abnor- mal Facies; Mental Retarda- tion	Familial Study		Sex and Age	Chrom- osome Study	Intelli- gence	Systolic Injection Murmur Grade	Mild Micro- cephaly	Growth Failure	Surgery	
							Physical Findings	Cardiac Catheter- ization; Cardioan- giography								
D.McC.	A.III-14	N	N	N	N	o	+	+	F 11	N	N	IV	o	o	+	
B.McC.	A.III-16	↑	LV ↑	N	N	o	+	+	M 4	N	N	IV	o	o	+	
S.McC.	A.III-18	N	N	N	N	o	+	+	F 3	N	N	II	o	o	o	
C.K.	A.III-32	N	N	N	N	o	+	+	M 4	N	N	IV	o	o	o	
C.H.	A.III-33	N	N	↓	N	o	+	+	M 13	N	N	V	o	o	+	
J.B.	B.II-1	N	RA ↑	↓	N	o	+	+	M 12	N	N	V	o	o	+	
I.B.	B.II-2	N	N	N	N	o	+	+	M 10	N	N	IV	o	o	o	
C.	C.II-1	N	N	N	N	o	+	+	F 15	N	N	IV	o	o	o	
J.C.	C.II-2	N	N	N	N	o	+	+	F 13	N	N	III	o	o	o	
K.O.J.	D.I-6	N	N	N	N	o	+	+	M 30	N	N	III	o	o	o	
O.J.	D.II-3	N	N	N	N	o	+	+	F 4	N	N	III	o	o	o	
R.A.	H.IV-2	↑	LV ↑	↓	↑	+	+	+	M 9	...	Retarded	V	+	+	+	
L.A.	H.IV-3	N	N	N	N	+	+	+	F 8	...	Retarded	IV	+	o	o	
M.L.	I.II-2	↑	LA LV ↑	↓	N	+	o	...	M 18	N*	Retarded	VI	+	+	+	
F.A.	J.II-1	N	N	N	N	+	o	...	F 3	N	Retarded	III	+	+	o	
R.W.	K.II-1	N	RA ↑	N	N	+	o	o	M 6	N	Retarded	III	+	+	o	
O.B.	L.II-3	↑	LA LV ↑	↓	N	+	o	...	M 16	N	Retarded	IV	+	+	+	
R.S.	M.II-2	↑	RA LV ↑ LV ↑	↓	N	+	o	...	M 11	N	Retarded	V	o	+	+	
S.H.	S.II-2	N	N	N	N	o	o	...	M 6	N	N	IV	o	+	+	
K.N.	T.II-4	↑	LV ↑	↑	N	o	o	...	M 13	N	N	V	o	o	+	
M.B.	U.II-1	↑	LV ↑	↑	N	o	o	...	M 16	...	N	IV	o	o	+	
G.F.	R.II-4	N	N	N	↑	o	o	...	F 6	N	N	V	o	o	o	
T.P.	N.II-2	↑	RV ↑	N	↑	+	o	...	F 24	...	Retarded	III	+	+	o	
R.M.	V.II-2	N	N	↑	N	o	o	...	F 10	...	N	V	o	o	o	
O.T.	O.II-2	N	N	↓	N	+	o	...	F 10	...	Retarded	IV	+	o	o	
K.W.	G.II-5	N	N	↓	N	o	+	...	M 5	...	N	V	o	o	o	
L.B.	F.III-2	N	N	↓	N	o	+	o	M 14	...	N	VI	o	o	o	

Classification		Cardioangiographic Data											Cardiac Catheterization Data		
		Ascending Aortic Length	Sinus of Val-salva Size	Aortic Valve Appearance	Coro-nary Artery Size	Other Cardio-vascular Defects	Ductus Diverti-culum	Aortic Insuffi-ciency	Brachio-cephalic Trunk	Left Ventric-ular Cavity Size	Left Ventric-ular Wall Size	Aortic Pulsa-tion	Resting Intrapul-monary Artery Systolic Gradient (mm. Hg)	Resting Left Ventric-ular Aortic Systolic Gradient (mm. Hg)	Post Isopro-terenol Left Ventric-ular Aortic Systolic Gradient (mm. Hg)
1a	III	↓	2+	N	2+	o	+	o	N	N	2+	↓	o	77	...
1a	III	↓	3+	N	2+	o	o	o	N	N	2+	↓	M.P.A. systolic pressure = 76	64	...
1b	III	N	2+	N	2+	o	o	o	N	N	N	N	62	25	...
1b	1b	N	2+	N	1+	o	o	o	N	1+	N	↑	34	34	78
1a	o	↓	3+	N	3+	o	o	o	Dilated innom.	1+	3+	↓	10	78	195
1a	III	↓	3+	N	2+	Aortic coarctation	o	o	Stenosis L.C.C.	1+	2+	↓	20	150	...
1b	1b	N	2+	N	2+	o	o	o	N	N	1+	↑	10	28	138
1b	III	N	2+	N	2+	o	o	o	...	N	N	N	16	21	70
1b	o	N	N	N	1+	o	o	o	N	N	N	N	16	17	66
1b	III	N	2+	Abnormal rt. cusp	2+	o	+	2+	...	1+	1+	↑	30	o	...
1b	1a	N	N	N	N	Aortic coarctation	o	o	N	N	N	↑	3	3	17
1a	III	↓	3+	N	3+	Aortic coarctation	o	o	Dilated innom.	2+	2+	↓	64	82	...
1b	III	N	N	N	N	Aortic coarctation	o	o	Retroesoph-ageal R.S.A.	N	N	↑	15	o	45
1a	o	↓	3+	N	4+	Absent lt. renal artery	o	o	Stenosis R.C.C., L.C.C. and L.S.A.	1+	3+	↓	o	171	...
1b	III	N	2+	N	2+	o	o	o	Common origin innom. and L.C.C.	2+	N	↑	8	14	22
1b	III	N	2+	N	1+	o	o	o	N	N	N	↑	35	15	...
1a	o	↓	4+	N	4+	o	o	o	Stenosis innom. at bifurcation	2+	3+	↓	11	195	...
1a	III	↓	4+	N	2+	o	o	o	Stenosis innom.	N	3+	↓	o	156	...
1a	o	↓	2+	Thick	1+	Ventric-ular septal defect	+	o	Dilated innom.	1+	3+	↓	...	81	...
II	...	N	Shallow	Thick	3+	o	o	o	N	N	4+	↑	o	183	...
II	...	N	Shallow	Thick	3+	o	o	o	N	N	3+	↑	o	95	...
1b	1a	N	1+	N	1+	Atrial septal defect	o	1+	Stenosis L.S.A.	1+	N	N	o	59	100
1b	III	N	2+	N	1+	Bilateral renal artery stenosis	o	o	Stenosis innom. with proximal and distal dilatation	N	N	↑	M.P.A. systolic pressure = 83	21	...
II	o	N	Shallow	Thick	N	Aortic coarctation	o	1+	Dilated innom. and L.C.C.	N	2+	↑	...	...	...
1b	1a	N	3+	Abnormal rt. cusp	1+	o	o	o	N	N	1+	↑	11	41	...
1b	1b	N	2+	N	2+	o	o	o	N	1+	N	↑	15	68	...
1a	o	↓	3+	N	3+	o	+	2+	Stenosis R.C.C.	N	3+	↓	9	157	...

\* Initial chromosome study indicated 46/47 mosaic with an extra chromosome resembling the 19-20 group. Subsequent study disclosed normal number with apparently normal karyotype.

KEY-

N = normal

↑ above normal

↓ below normal

LV = left ventricle

LA = left atrium

RV = right ventricle

RA = right atrium

innom. = innominate

L.C.C. = left common carotid

R.C.C. = right common carotid

L.S.A. = left subclavian artery

M.P.A. = main pulmonary artery

SVAS = supra-valvar aortic stenosis

PPAS = post-valvar pulmonary artery stenosis



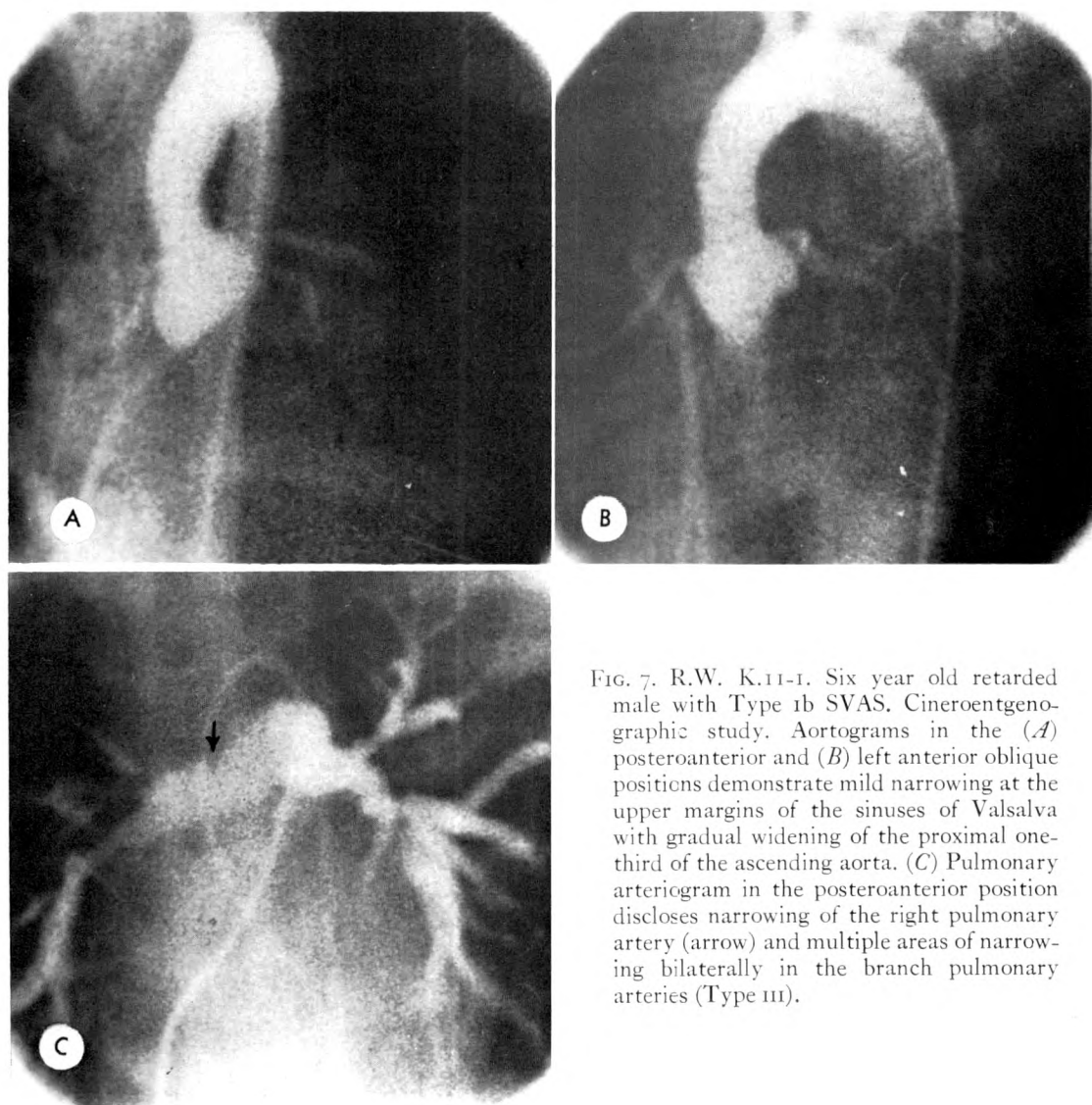


FIG. 7. R.W. K.11-1. Six year old retarded male with Type 1b SVAS. Cinerentgenographic study. Aortograms in the (A) posteroanterior and (B) left anterior oblique positions demonstrate mild narrowing at the upper margins of the sinuses of Valsalva with gradual widening of the proximal one-third of the ascending aorta. (C) Pulmonary arteriogram in the posteroanterior position discloses narrowing of the right pulmonary artery (arrow) and multiple areas of narrowing bilaterally in the branch pulmonary arteries (Type III).

Five of the 13 cases had Type 1a SVAS and 8 had Type 1b. Ten of the 13 had postvalvar pulmonary artery stenosis.

Genetic studies suggest that Type 1 SVAS is transmitted as an autosomal dominant with variable penetrance.<sup>18</sup>

*Sporadic SVAS with normal intellect.* This group includes cases without familial evidence of SVAS, facial peculiarities, or mental retardation. Two cases of this description were encountered in our series (exclusive of Type II SVAS which will be discussed more fully later). One with Type 1a SVAS had a ventricular septal defect and the other with Type 1b had an atrial septal

defect. The former also had postvalvar pulmonary artery stenosis.

*SVAS and Marfan's syndrome.* This association has been reported in at least 3 patients, one a 12 year old child.<sup>5,8,19</sup> We did not encounter a case with Marfan's syndrome.

#### DISCUSSION

The angiographic diagnosis of mild SVAS with Type 1b may be extremely difficult. Only when one realizes that supravulvar narrowing occurs in a spectrum from minimal to very severe can one expect to recognize the milder cases. In 9 of 14

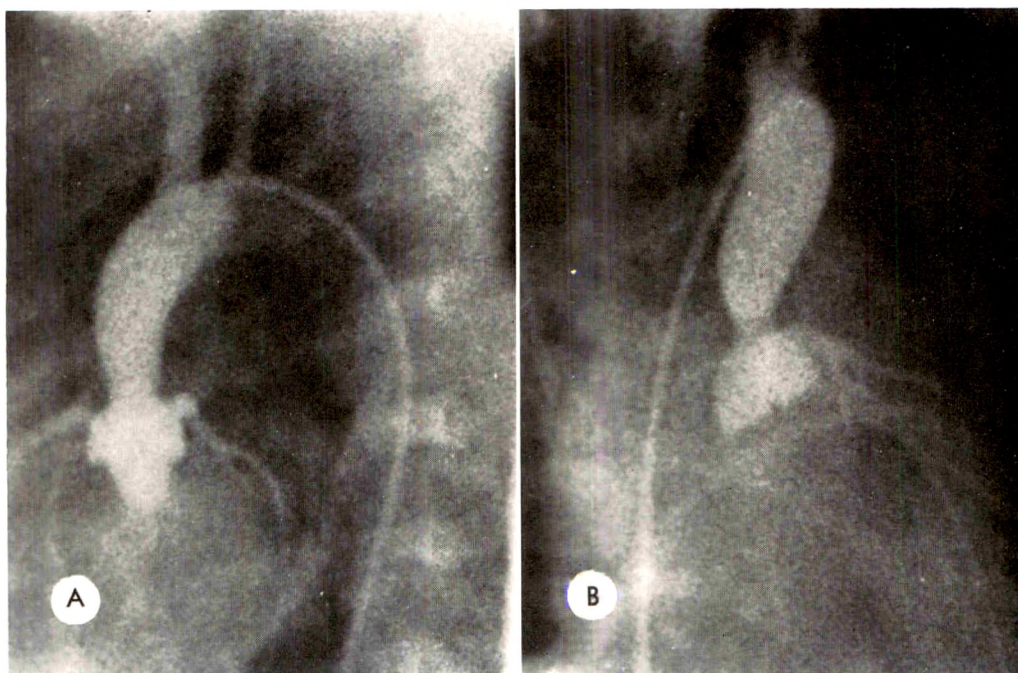


FIG. 8. J.W. G.11-5. Five year old male with normal intellect and Type 1b SVAS. Cinerentgenographic study. Aortograms in the (A) left anterior and (B) right anterior oblique positions demonstrate maximal narrowing at the upper margins of the sinuses of Valsalva with gradual widening of the proximal one-third of the ascending aorta. The narrowing is more marked in the right anterior oblique position, a frequent finding.

cases with Type 1b SVAS, the resting left ventricular-aortic systolic pressure gradient was 25 mm. Hg or less. Isoproterenol was used in several of these milder cases to increase the gradient and permit localization of the site of stenosis by withdrawal of an end-hole catheter from the left ventricle to the aortic arch. Peculiar facies, mental retardation, dental anomalies, and postvalvar pulmonary artery stenosis occurred as often with mild SVAS as with severe.

Type II SVAS, as defined in this discussion, is different in many ways from the other forms of supravalvar narrowing. In contrast to Type I, the sinuses of Valsalva are shallow and not dilated. During systole, the thick aortic valve has the appearance of a dome. The mid and distal ascending aorta is moderately to markedly dilated. Both of these features are seen in aortic valve stenosis. The supravalvar narrowing, however, is more marked than that noted in any of 40 cinecardioangiograms reviewed of patients with typical aortic valve

stenosis. Although it is possible that the aortic valve cusps undergo thickening as a result of the trauma of repeated opening against the supravalvar narrowing, it seems more likely that the abnormal valve cusps, small annulus, and narrow supravalvar aortic segment are part of the same anomaly. No patient with Type II SVAS had evidence of postvalvar pulmonary artery stenosis, mental retardation, or familial evidence of congenital heart disease.

Two cases with Type II SVAS were found to have thick semirigid valves without commissural fusion at the time of surgical exploration. This precluded aortic valve commissurotomy. It is obvious from postoperative cardiac catheterization of these cases that relief of the supravalvar obstruction alone will not adequately resolve the obstruction. Replacement of the aortic valve may also be necessary. None of our patients with Type 1b SVAS has required surgical treatment. The extreme length of



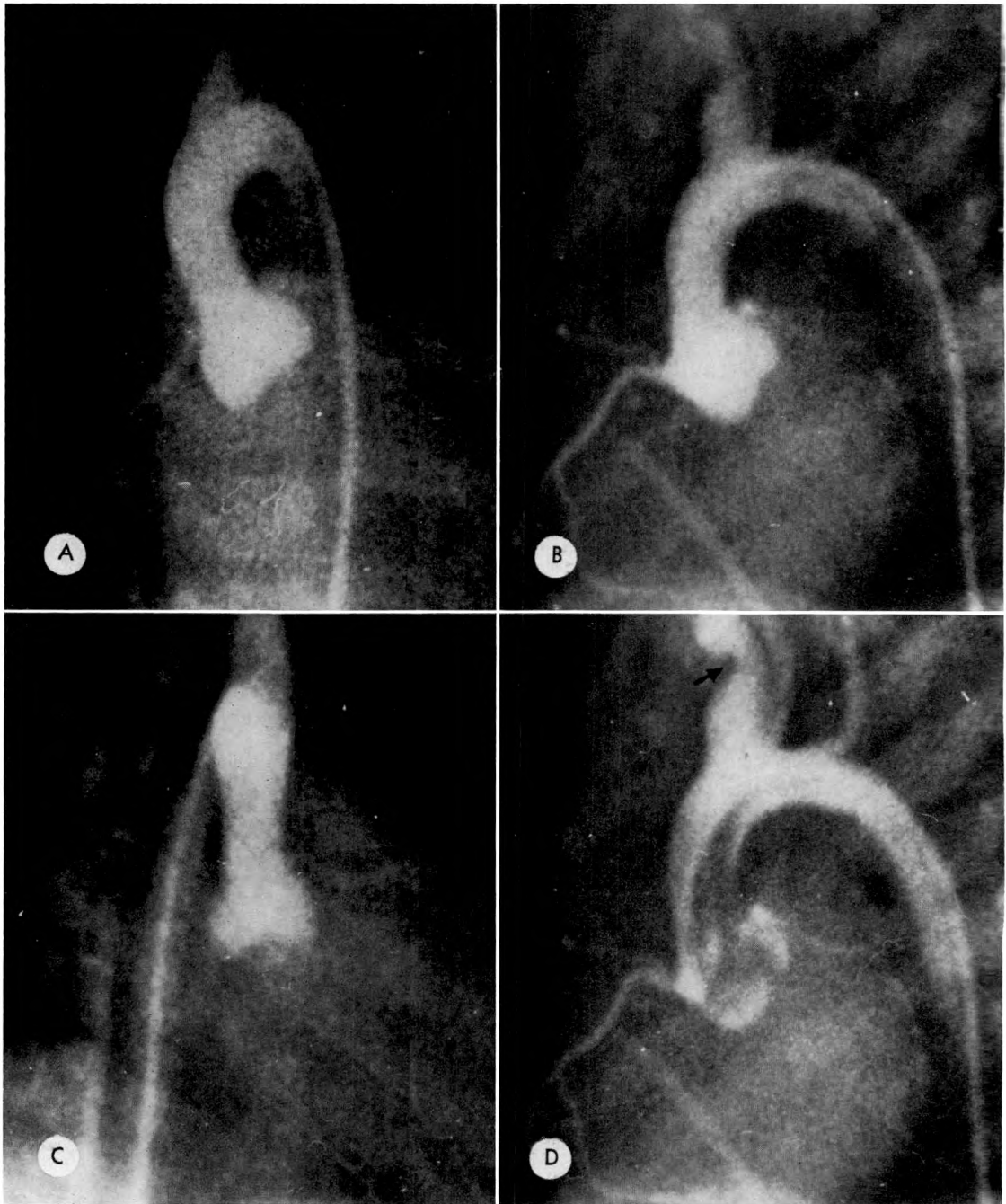


FIG. 9. T.P. N.11-2. Two and one-half year old retarded female with Type 1b SVAS. Cineroentgenographic study. Aortograms in the (A) posteroanterior, (B) left anterior oblique and (C) right anterior oblique positions show mild narrowing of the proximal one-third of the ascending aorta beginning at the upper margins of dilated sinuses of Valsalva. (D) Aortogram in the left anterior oblique position demonstrates the unopacified jet of left ventricular blood entering the aorta. Note the dilated innominate artery below and above an area of narrowing (arrow).

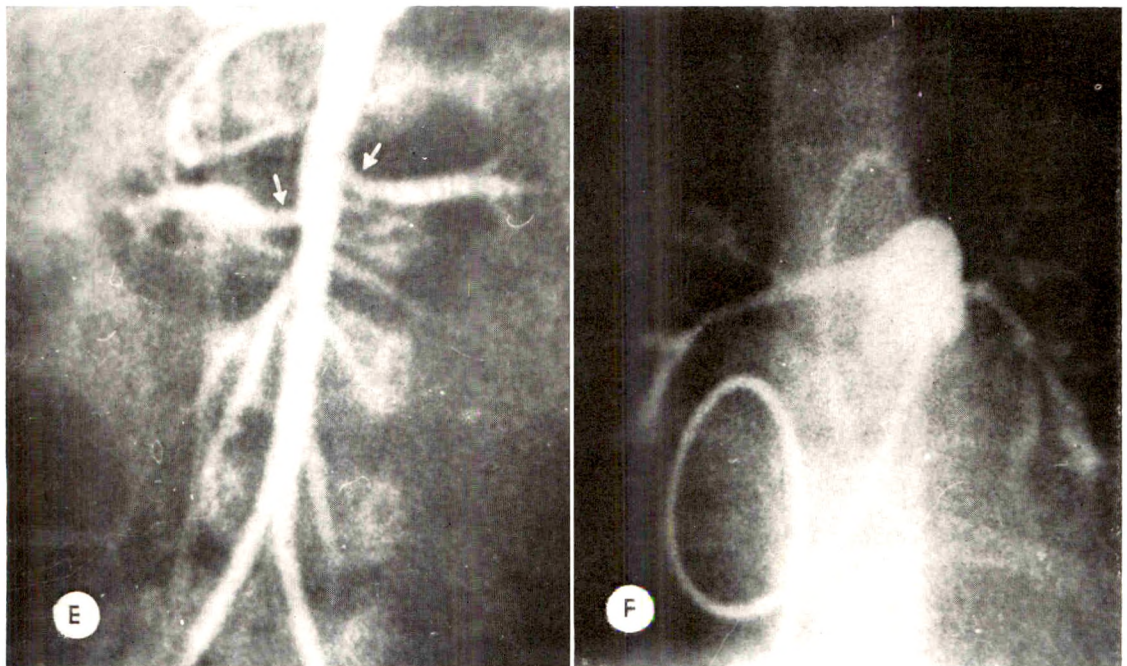


FIG. 9. (E) Abdominal aortogram shows bilateral renal artery narrowing (arrows). (F) Pulmonary arteriogram in the posteroanterior position demonstrates diffuse narrowing of the right and left pulmonary arteries as well as multiple areas of narrowing bilaterally in the branch pulmonary arteries (Type III).

Type 1a SVAS requires aortotomy and insertion of a gusset if obstruction is to be effectively relieved.

Unless pulmonary arteriograms are made in frontal and oblique projections, the diagnosis of localized pulmonary artery stenosis in some cases may be overlooked. This is so because the bifurcation of the main pulmonary artery is partly obscured by the main pulmonary artery itself in the frontal projection and, in addition, peripheral pulmonary arteries oriented in a sagittal plane cannot be seen clearly if only a frontal plane angiogram is obtained. Pulmonary cinearteriograms of good quality permit detection of mild degrees of narrowing by appreciation of diminished systolic expansion. It should be noted that a pulmonary artery of normal internal diameter does not necessarily have a wall of normal thickness. Case D.E.—L.II-3 (Table 1) had a normal pulmonary arteriogram but was found at the time of surgery by palpation to have marked thickening of the wall of the main pulmonary trunk and

of the proximal one-third of the left pulmonary artery.

Nearly half of the cases of Type 1 SVAS had palpable asymmetry of the brachial pulses with about one-third manifesting asymmetry of the carotid pulses. Upper extremity blood pressure differences exceeding 12 mm. Hg were noted frequently. In some instances, these findings were correlated with narrowing of a brachiocephalic trunk as determined by aortography. In others, this correlation was absent. It is speculated that blood ejected from the left ventricle is directed in a jet by the supravalvar aortic stenosis into a brachiocephalic trunk, most often the innominate, with resultant asymmetry of brachial and carotid pulses.<sup>16,17</sup> Substantial dilatation of the innominate artery in 2 of our cases lends support to this theory.

Systolic ejection murmurs were heard in every case of SVAS included in this report. The intensity of these murmurs varied from Grade II to Grade VI. They were heard best in the suprasternal notch in contrast to the



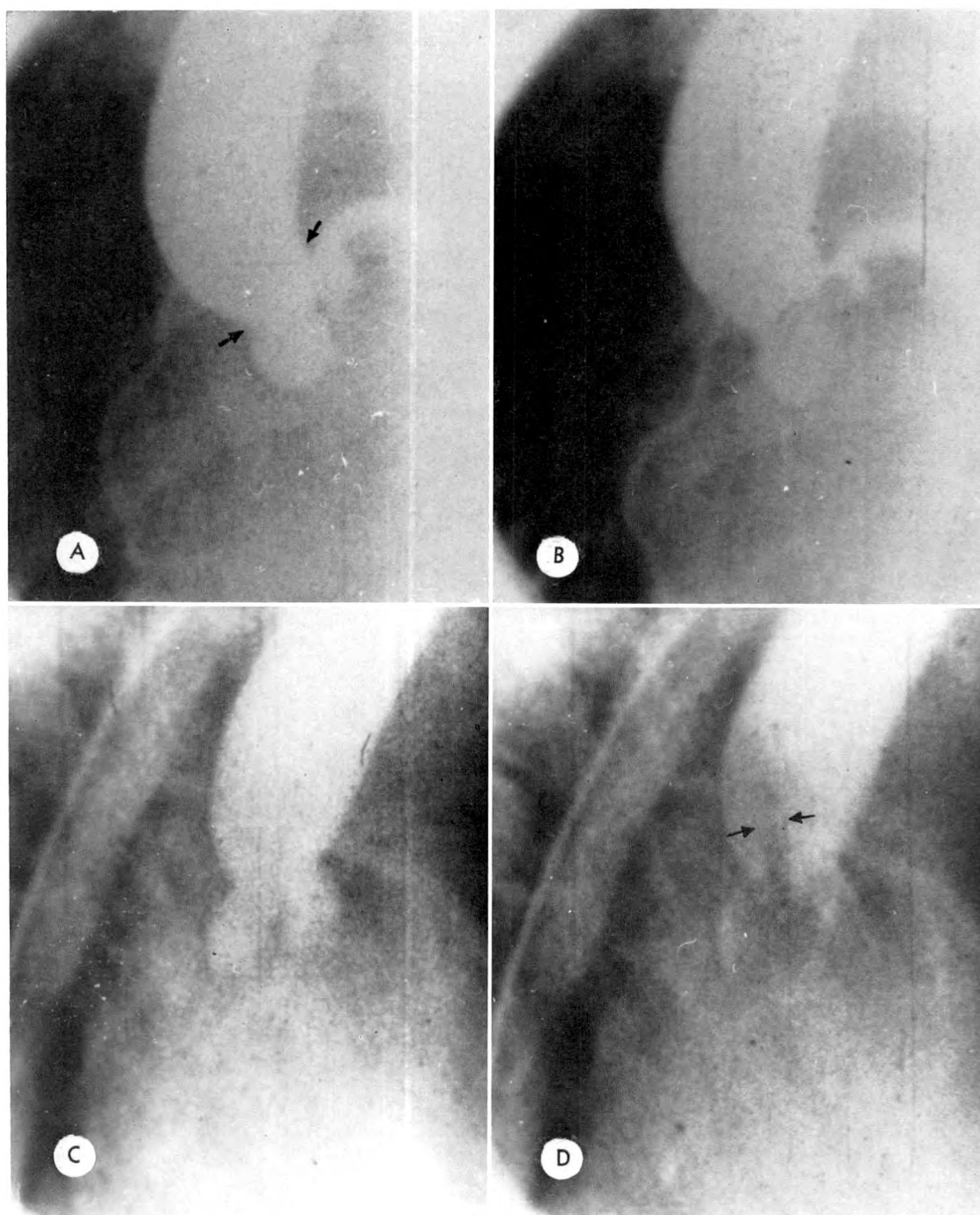


FIG. 10. K.N. T.11-4. Thirteen year old male, Type II SVAS. Cinerentgenographic study. Aortograms in the left anterior oblique position in (A) diastole and (B) systole show moderate narrowing (arrows) above shallow sinuses of Valsalva. Distal to this area the aorta is dilated. Aortograms in the right anterior oblique position in (C) diastole and (D) systole show moderate supravulvar narrowing. The aortic valve annulus is small and the commissures are unfused (surgical confirmation). Dome and jet (arrows) formation are shown in D.

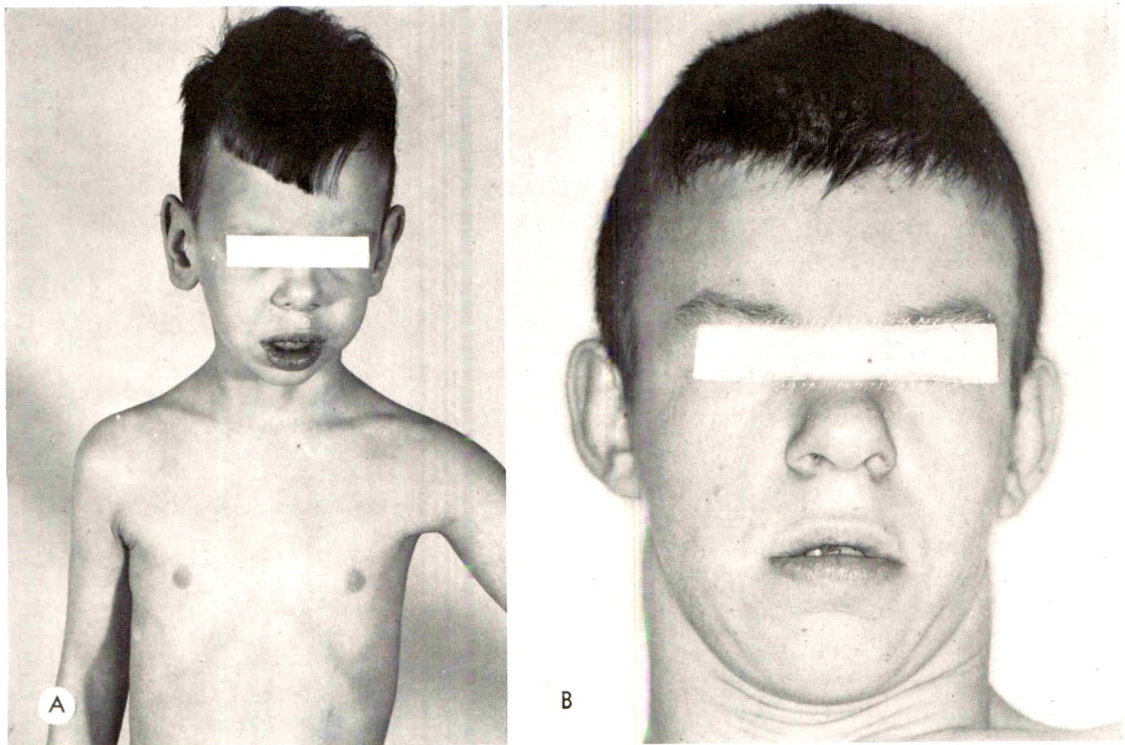


FIG. 11. (A) R.W. K. II-1. Six year old male. (B) D.E. L. II-3. Sixteen year old male. Both have the syndrome of SVAS, mental retardation, and peculiar facies. Note thick pouting upper lips, wide nasal philtrums, depressed nasal bridges and small mandibles. Macrocornea, a feature of idiopathic infantile hypercalcemia, was not present in these patients.

findings in aortic valve stenosis where they are usually loudest at the cardiac base. The systolic ejection click characteristic of aortic valve stenosis was absent in all patients with Type I SVAS.

No case in this series developed congestive heart failure from aortic obstruction but 2 had bacterial endarteritis.

#### SUMMARY

The roentgen experience with 27 patients with supravalvar aortic stenosis (SVAS) who had both cardiac catheterization and cinecardioangiography is presented.

Gross and microscopic pathologic features of SVAS in previously reported cases are reviewed. Observations at the time of surgery in 11 cases of this series are detailed.

A classification of SVAS and postvalvar pulmonary artery stenosis (PPAS) is sug-

gested based on the cardioangiographic findings in these 27 cases. One form of SVAS (Type II), heretofore not emphasized, is a complex anomaly with mild to moderate supravalvar narrowing just above shallow sinuses of Valsalva, small aortic valve annulus, and thick semirigid valve cusps with unfused commissures.

Conditions with which SVAS is associated are briefly discussed. These conditions include: the syndrome of SVAS, mental retardation and peculiar facies, idiopathic infantile hypercalcemia, familial SVAS with normal intellect, sporadic SVAS with normal intellect, and Marfan's syndrome.

The basic clinical and hemodynamic characteristics of SVAS are described and a table is presented summarizing plain film, clinical, cardioangiographic and cardiac catheterization data in every case in this series.



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## ADDENDUM

Since the completion of this manuscript, a few cases of familial idiopathic hypercalcemia have come to our attention. Fraser *et al.* (FRASER, D., KIDD, B.S.L., KOOH, S. W., and PAUNIER, L. A new look at infantile hypercalcemia. *Pediat. Clin. N. America*, 1966, 13, 503-525) have recently reviewed the subject of idiopathic hypercalcemia and present evidence favoring the fact that SVAS, mental retardation and peculiar facies is the late normocalcemic stage of severe infantile hypercalcemia. Further evidence will be necessary to conclusively prove this association.

Twelve of thirteen cases which formed the basis for an Exhibit on Supravalvar Aortic Stenosis at the 1963 meeting of the American Roentgen Ray Society are included in this paper. The thirteenth case, not included here, was from the University of Oklahoma School of Medicine.

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## CONGENITAL AORTIC STENOSIS\*

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**S**URGICAL treatment of all types of congenital aortic stenosis is now possible, and such treatment is indicated for the majority of patients having this condition who have significant obstruction to left-ventricular outflow. Although cardiac catheterization data provide vital information in the study of patients with congenital aortic stenosis, angiocardiology is of great value in determining the exact location and nature of the obstructive process as well as in detecting the presence and pathologic anatomy of associated anomalies of the heart and great vessels.

The purpose of this study was to evaluate the roentgenologic findings in a series of cases of congenital aortic stenosis, valvular, supraventricular, and subvalvular. Particularly, we wanted to evaluate and stress the importance of angiocardiology in this group of conditions.

### MATERIAL AND METHODS

From the files of Cardiovascular Radiology of the Mayo Clinic, all records were reviewed of cases from the 4 years from April 1, 1960, through April 1, 1964, in which the angiocardiology diagnosis of aortic stenosis had been made. In an effort to include only cases of congenital aortic stenosis, all cases in which acquired valvular stenosis was thought to be present at operation or autopsy and all cases of valvular stenosis in which a history of rheumatic heart disease or bacterial endocarditis was elicited were excluded.

Of the 40 cases selected for study, 23 had isolated valvular aortic stenosis, 7 had isolated supraventricular aortic stenosis, and 7 had isolated subvalvular aortic stenosis. One patient had combined valvular and

supraventricular aortic stenosis, and 2 had combined valvular and subvalvular stenosis (Table 1).

Roentgenograms of the chest and angiocardiology were available for review in each case, and in all instances measurements of the pressure gradient across the outflow region of the left ventricle had been obtained. Surgical treatment had been carried out in 6 of the 23 cases of isolated valvular stenosis, in 4 of the 7 cases of isolated supraventricular stenosis, and in 5 of the 7 cases of isolated subvalvular stenosis. Operation had also been performed in the 2 cases of combined valvular and subvalvular aortic stenosis. The surgical records on all operated patients were reviewed and the findings at operation were compared with the angiocardiology findings.

The angiocardiology in all instances consisted of large-film biplane examinations, carried out with injection of contrast medium into the left ventricle, aortic root, or pulmonary artery and filming at the rate of 6 to 12 films/sec. during the phase of left heart opacification.

### VALVULAR AORTIC STENOSIS

*Morphologic Features.* Two varieties of congenital valvular aortic stenosis are generally recognized.<sup>13</sup> The most common type is simple dome-shaped stenosis, in which the aortic valve is a fibrotically thickened structure in the shape of a dome, the apex of which is directed toward the distal aortic lumen. The size of the opening at the apex of the dome is variable. Usually, three raphe, which appear to represent abortive commissures, may be identified at the inferior portion (base) of the dome-like valve (Fig. 1). Occasionally, only two such

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TABLE I  
CONGENITAL AORTIC STENOSIS, ISOLATED AND COMBINED FORMS

Type of Aortic Stenosis	Patients		Age		Sex		Angio-grams	Opera-tions
	No.	Per Cent	Range	Average	M	F		
Isolated valvular	23	57.5	2 mo. to 31 yr.	10 yr.	19	4	23	6
Isolated supra-valvular	7	17.5	6 to 21 yr.	10 yr.	4	3	7	4
Isolated subvalvular	7	17.5	13 to 45 yr.	29.5 yr.	3	4	7	5
Combined supra-valvular and valvular	1	2.5	5 wk.	5 wk.	0	1	1	0
Combined valvular and subvalvular	2	5	7 to 9 yr.	8 yr.	1	1	2	2
Total	40	100	5 wk. to 45 yr.	13.6 yr.	27	13	40	17

raphes exist, in which case the valve may be viewed as the equivalent of a bicuspid valve.

The second type is a unicommissural, dome stenosis, which is less common. The aortic valve is represented by a single flap of fibrotically thickened valvular tissue which extends around the aortic wall, beginning and ending at the single commissure (Fig. 2). In both types, the aortic valve is, in addition, often insufficient. Left ventricular hypertrophy and endocardial sclerosis of the left ventricular outflow tract are usually present, as in other types of aortic stenosis.

In addition to these two common types, the congenitally bicuspid valve theoretically may be stenotic without superimposed valvular disease. However, in most cases of congenital bicuspid aortic valve, stenosis of the valve is acquired as a result of con-

tinuous trauma or superimposed bacterial endocarditis.

*Cases Studied.* There were 26 patients, 20 male and 6 female, who had congenital valvular aortic stenosis (Table 1). Their ages ranged from 5 weeks to 31 years. The following associated cardiac lesions were observed: mitral insufficiency (3 cases), aortic insufficiency (5 cases), supra-valvular aortic stenosis (1 case), subvalvular aortic stenosis (2 cases), coarctation of the aorta (1 case), and large patent ductus arteriosus (1 case).

*Roentgenographic Findings.* The cardiothoracic index ranged from 0.37 to 0.69, the average being 0.50. The left ventricle was considered to be enlarged in 21 cases (81 per cent) and was of normal size in 5. This



FIG. 1. Usual form of dome-shaped valvular aortic stenosis viewed from above.



FIG. 2. Aortic (left) and pulmonary (right) valves viewed from above. Aortic valve shows unicommissural dome-shaped valvular aortic stenosis.



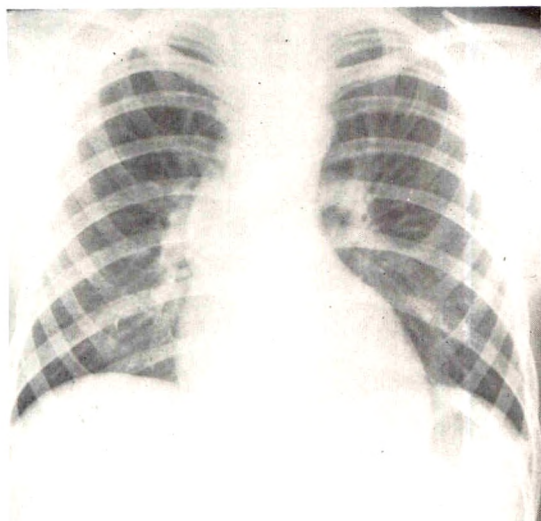


FIG. 3. Posteroanterior roentgenogram of chest of patient having valvular aortic stenosis. Cardiothoracic index=0.51. There are Grade 2 (on the basis of 1 to 3) enlargement of the left ventricle and Grade 3 poststenotic dilatation of the ascending aorta.

enlargement was slight in 11 cases, moderate in 7, and severe in 3. The left atrium was normal in size in 24 cases, while in 2 cases it was slightly enlarged.

Poststenotic dilatation of the ascending aorta was observed in 9 (35 per cent) of the 26 cases (Fig. 3). The poststenotic dilatation was slight in 4 cases, moderate in 4 cases, and marked in 1 case.

The pulmonary vasculature was normal in all cases except 1, in which there was an associated large patent ductus arteriosus.

None of the 26 patients having valvular aortic stenosis exhibited valvular calcification on fluoroscopic or roentgenographic examination.

*Angiocardiography. Technique.* Angiocardiography was carried out in 19 cases by injection of contrast medium into the left ventricle, into which a catheter had been passed from the axillary artery in retrograde fashion through the aortic valve. In 3 instances in which the catheter could not be passed through the aortic valve, the injection was made into the main pulmonary artery with rapid serial filming during the phase of left heart opacification. In 6 in-

stances, injection was made into the aortic root, and in 1 instance a left ventriculogram was obtained after percutaneous trans-thoracic left-ventricular needle puncture.

*Observations.* The aortic valve was tricuspid in 23 cases, and the number of cusps was indeterminate in 3 cases. Thickening and irregularity of the aortic valve leaflets were apparent in 22 cases (Fig. 4 through 7). In 3 cases, the valve leaflets appeared irregular but unthickened, while in 1 case the valve leaflets appeared slightly thickened without any evidence of irregularity of the leaflets.

The aortic valve was noted to be definitely dome-shaped during systole in 20 of the 26 cases. In 5 cases, doming of the aortic valve was not observed, and in 1 case the aortic valve was rigid and showed no appreciable motion between systole and diastole.

The maximal diameter of the aperture in the aortic valve could be accurately measured in most cases on the angiocardiographic films exposed during ventricular systole. The diameter of the aperture varied from 3 mm. to 25 mm., the average being 13 mm. Although there was a tendency for the higher pressure gradients across the valve to be associated with the smaller apertures, there was no close correlation between the pressure gradient and the diameter of the aperture as determined from the angiocardiogram.

A distinct positive or negative jet emanating from the aperture in the aortic valve was observed in 9 of the 26 cases.

Poststenotic dilatation of the ascending aorta could be evaluated accurately on the angiocardiograms and was observed in 21 (81 per cent) of the 26 cases. The poststenotic dilatation was considered to be slight in 6 cases, moderate in 7, and marked in 8. There was no distinct correlation between the severity of the valvular stenosis, as determined by the pressure gradient, and the degree of poststenotic dilatation.

The size of the left ventricular chamber was normal in 6 cases, slightly enlarged in 11, and moderately to markedly enlarged



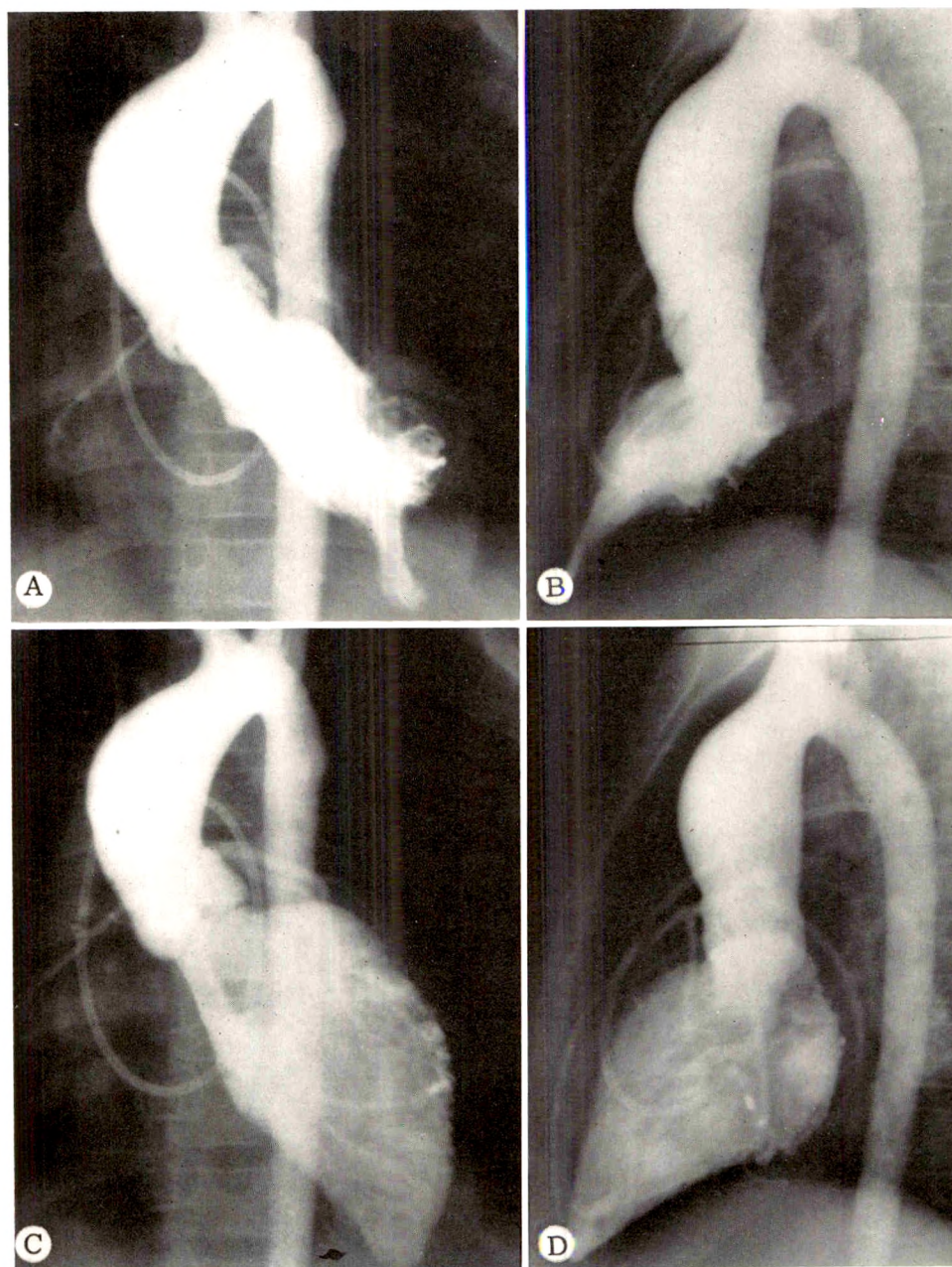


FIG. 4. Valvular aortic stenosis in a 6 year old boy with gradient of 30 mm. Hg across valve. (A and B) Systole, frontal and lateral. (C and D) Diastole, frontal and lateral. Aortic valve leaflets are irregularly thickened and are dome-shaped in systole. There are marked poststenotic dilatation of the ascending aorta and left ventricular enlargement and hypertrophy.

in 8. In 1 case the left ventricular chamber was considered to be smaller than normal.

Concentric hypertrophy of the left ventricle, as determined from measurement of the thickness of the lateral wall of the left ventricle in diastole, was observed in 20

cases (77 per cent). In 6 cases (23 per cent), hypertrophy of the upper portion of the interventricular septum was present, and in 2 of these slight subvalvular narrowing of the left ventricular outflow tract resulted. Hypertrophy of the individual papillary



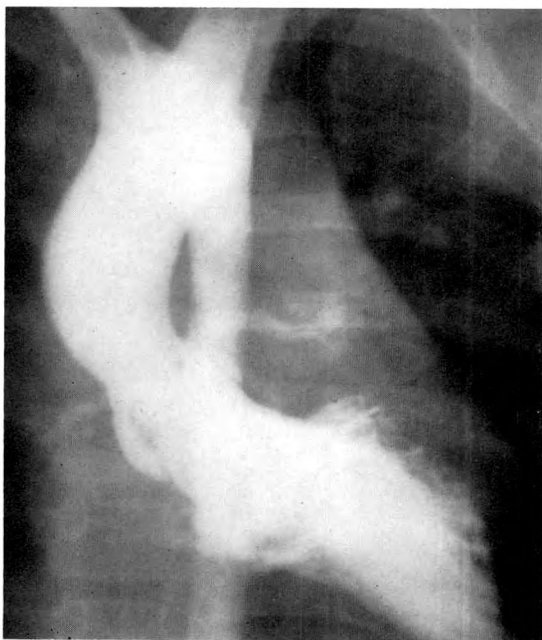


FIG. 5. Valvular aortic stenosis in an 11 year old boy having pressure gradient of 65 mm. Hg across valve. Frontal angiogram in systole shows thickened, irregular, dome-shaped aortic valve leaflets in systole. There are moderate post-stenotic dilatation of aorta and marked dilatation of each branch of aortic arch.

muscles of the left ventricle was evident in 11 cases (42 per cent).

Left ventricular function, as evaluated on the basis of contraction and emptying of the left ventricle in systole, was good in 25 cases and poor in 1 case.

The coronary arteries were of normal size in 24 cases; they could not be evaluated in 2 cases in which the study had been carried out by injection of contrast medium into the pulmonary artery.

Mitral insufficiency was evident in 3 of the 26 cases of valvular aortic stenosis, but it was mild in each instance. The competency of the aortic valve was evaluated in 6 cases by means of aortic-root injection, and in 5 of these, aortic insufficiency was present. The insufficiency was graded as mild in 3 cases and moderate in 2.

#### SUPRAVALVULAR AORTIC STENOSIS

*Morphologic Features.* Supravalvular stenosis, the rarest form of obstruction at or

near the aortic valve, is generally thought to be a congenital anomaly. The prototype example is one in which a fibrous membrane or ring completely or partially encircles the aorta immediately above the aortic cusps. Commonly, a band or bands of connective tissue form parts of such a ring; and, in some instances, such bands may extend across the lumen of the aorta. Occasionally, the supravalvular aortic narrowing appears to result from localized hypoplasia of the aorta, while in other cases the narrowing is due to fibrous or fibroelastic thickening of the aortic wall (Fig. 8). In the latter instances, trabeculations of the thickened aortic segment may present as diaphragm-like ridges of intima. While the aortic cusps are usually anatomically normal, an associated bicuspid aortic valve is not uncommon. Other valvular anomalies, especially of the mitral valve, may coexist. Rarely, instances of incomplete dissecting aneurysm or small healed dissecting aneurysm located just above the valve may produce obstruction to left ventricular outflow.



FIG. 6. Valvular aortic stenosis in a 5 year old girl. Pressure gradient across valve was 30 mm. Hg. Frontal projection in systole shows irregular thickening of aortic valve leaflets and relative rigidity of left coronary cusp.

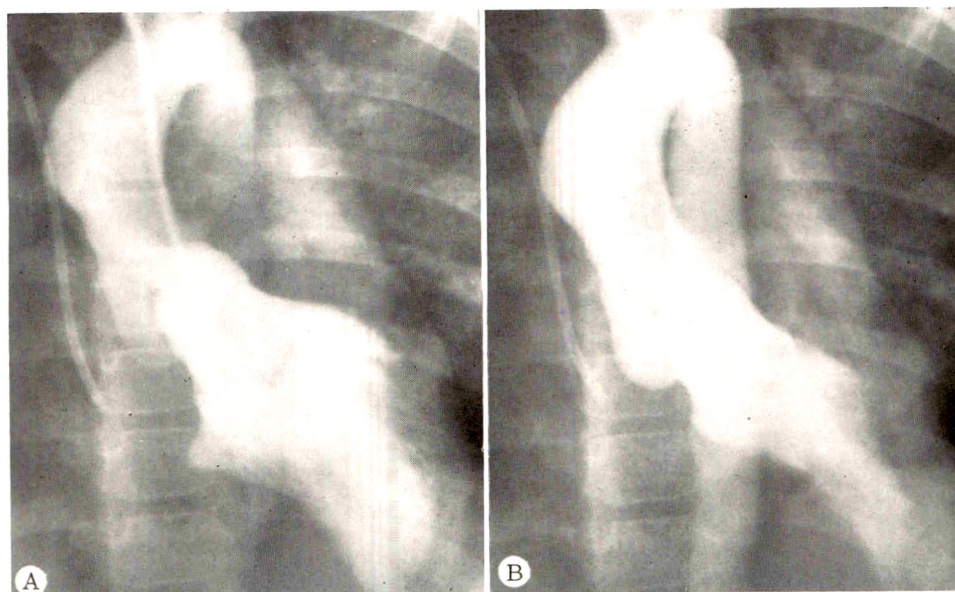


FIG. 7. Valvular aortic stenosis in a 6½ year old boy. Systolic pressure gradient across aortic valve was 36 mm. Hg. (A) Film in early systolic phase shows jet of contrast medium striking right lateral wall of aorta. (B) Film in later systolic phase shows marked poststenotic dilatation of ascending aorta. Orifice of aortic valve is slightly eccentric. Aortic valve leaflets are thickened and dome-shaped.



FIG. 8. One morphologic variety of supravalvular aortic stenosis. In this instance, narrowing resulted from fibroelastic thickening of the aortic wall above the valve associated with narrowing of aortic lumen (L). Left ventricular outflow tract

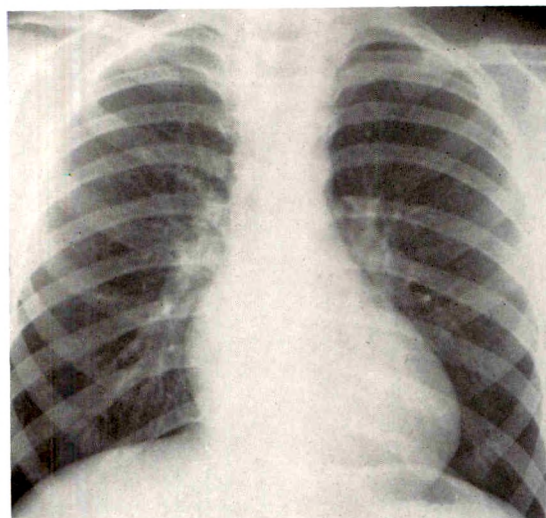


FIG. 9. Supravalvular aortic stenosis in a 7 year old boy. Posteroanterior roentgenogram of chest shows slight enlargement of left ventricle.

and aorta have been opened. There is post mortem distortion of aortic valve (arrow). Surgical repair of narrowed segment had been attempted.



*Cases Studied.* There were 8 patients, 4 male and 4 female, having supravulvular aortic stenosis, one of whom had associated valvular aortic stenosis (Table 1). For the 7 patients having isolated supravulvular aortic stenosis, the ages ranged from 6 years to 21 years and the average age was 10 years. The patient having coexistent

valvular aortic stenosis was a 5 week old girl.

*Roentgenographic Findings.* The cardiothoracic index ranged from 0.40 to 0.54, the average being 0.49. The left ventricle was of normal size in 6 cases and was slightly enlarged in 2 (Fig. 9). There was no evidence of left atrial enlargement in any of the 8

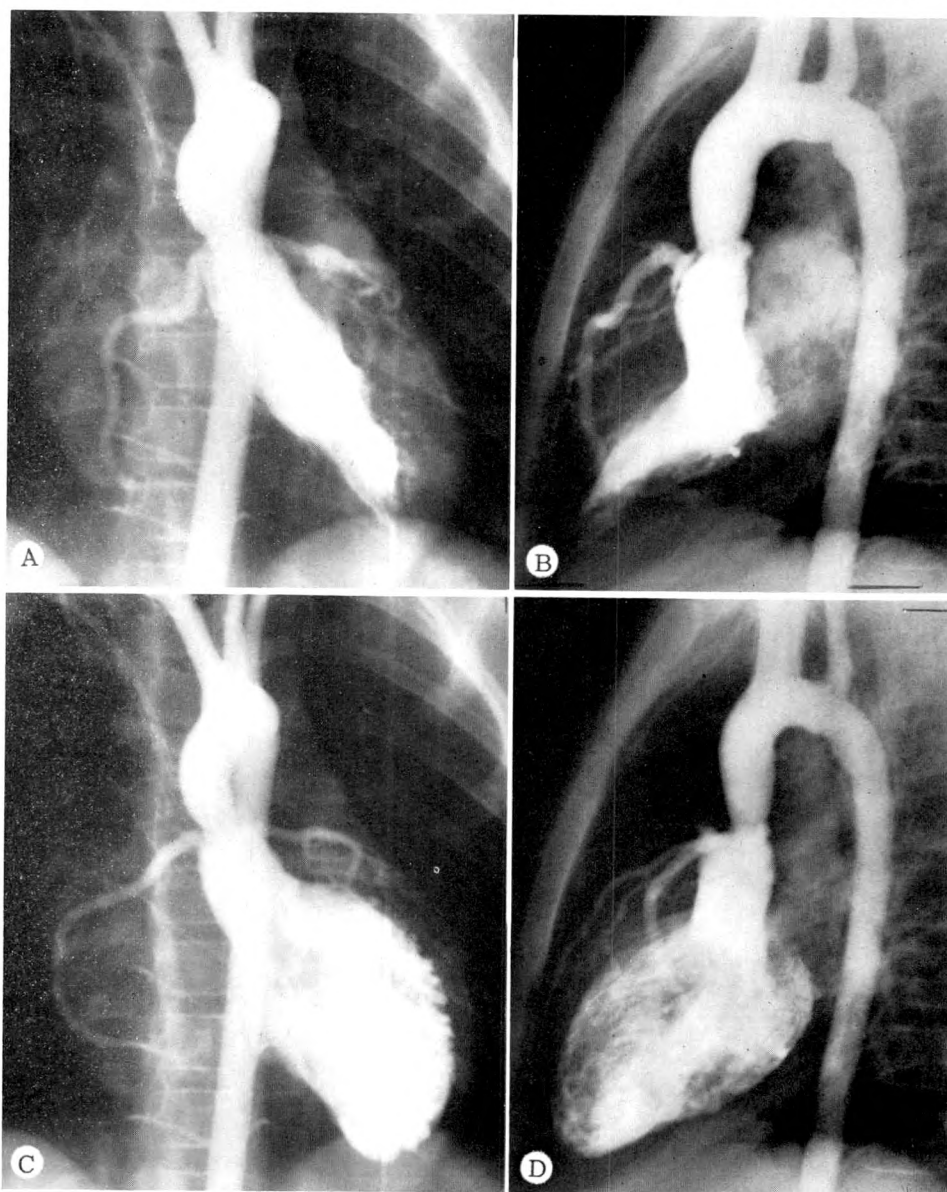


FIG. 10. Supravulvular aortic stenosis in a 6 year old girl. Gradient across stenotic zone was 57 mm. Hg. (A and B) Systole, frontal and lateral views. (C and D) Diastole, frontal and lateral views. Narrowed segment is located just above coronary ostia. Stenosis is more apparent in diastole. Right coronary artery is large. Mild mitral insufficiency is evident in lateral systolic film (B). (Patient also had mild infundibular pulmonary stenosis.)

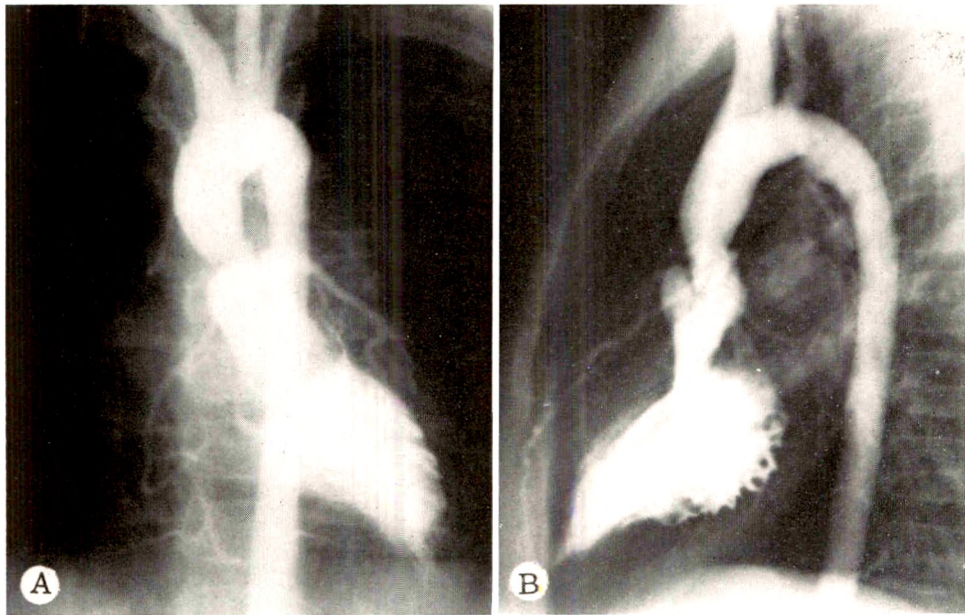


FIG. 11. Supravalvular aortic stenosis in a 6 year old girl with gradient of 60 mm. Hg across stenotic zone. (A and B) Frontal and lateral views in diastole show marked narrowing of ascending aorta just above orifices of coronary arteries. Narrowing is more marked on right anterior aspect of aortic root where diaphragm-like structure extends partially across aortic lumen from right anterior aspect of aortic wall.

cases. There was no roentgenographic evidence of poststenotic dilatation of the ascending aorta in any of the cases. The pulmonary vasculature was normal in all cases and in none was there evidence of intracardiac calcification.

**Angiocardiography. Technique.** Angiocardiography was carried out in 7 cases by means of injection into the left ventricle, into which a catheter had been passed from the axillary artery in retrograde fashion through the aortic valve. In 1 instance, a thoracic aortogram was carried out with injection into the aortic root.

**Observations.** In all 8 cases, the narrowing was located at or just distal to the aortic valve ring and involved the entire circumference of the aorta, although the narrowing was usually more marked on the right anterolateral aspect of the aorta (Fig. 10 through 12). In all cases the stenosed segment was located slightly distal to the ostia of the coronary arteries. The length of the stenosed segment varied from 3 to 7 mm. There was no correlation between the ratio of the diameter of the narrowed segment to

the diameter of the normal upper part of the descending aorta and the pressure gradient as determined by cardiac catheterization.

The sinuses of Valsalva were normal in 4 cases and were enlarged in 4 cases. The aortic valve was tricuspid in 6 cases, bicuspid in 1 case, and probably bicuspid in 1 case. The aortic cusps were thickened in 1 patient who had combined valvular and supravalvular stenosis (Fig. 13, A and B).

Poststenotic dilatation of the ascending aorta was evident on the angiocardiogram in 7 of the 8 cases. The dilatation was mild in 5 cases and moderate in 2. In no instance was marked poststenotic dilatation present. A jet was visible distal to the stenotic segment only in the case of combined valvular and supravalvular stenosis.

The coronary arteries were of normal size in 7 cases and were enlarged in 1 case.

The left ventricular chamber was slightly enlarged in 2 cases, of normal size in 3 cases, and slightly smaller than normal in 1 case. There was clear-cut evidence of left ventricular hypertrophy in only 1 case,



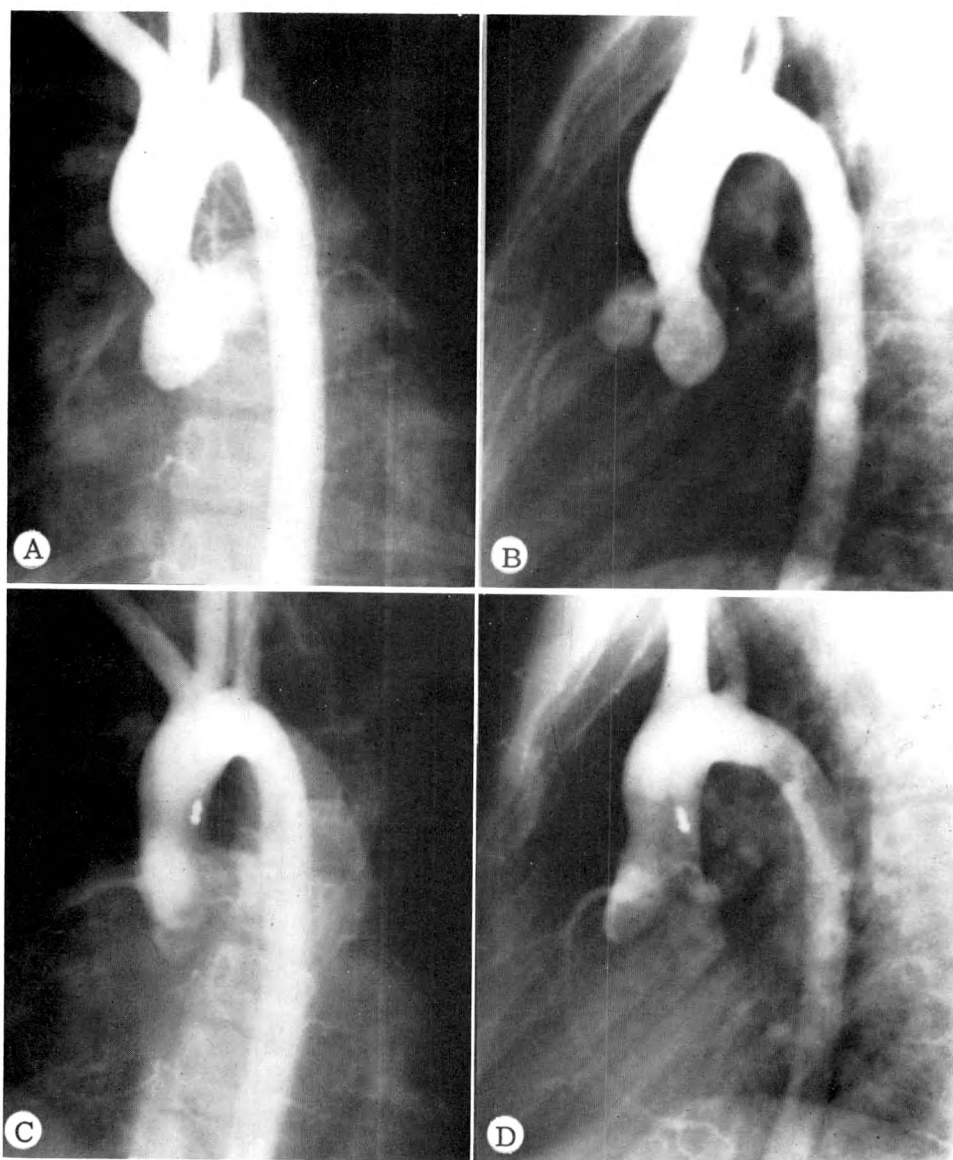


FIG. 12. (A and B) Preoperative thoracic aortogram of an 11 year old boy having supravulvar aortic stenosis. Gradient was 23 mm. Hg across region of stenosis. Entire circumference of ascending aorta just above aortic valve ring is narrowed, but narrowing is more pronounced on right anterolateral aspect. Sinuses of Valsalva are large. At time of operation, fibrous ring was found completely encircling aorta at level of commissures of aortic valve. Aperture through this region was estimated to be 4 mm. in diameter. (C and D) Postoperative thoracic aortogram shows relief of obstruction.

in which the left ventricular chamber was smaller than normal. The function of the left ventricle, as assessed angiographically with regard to contraction and emptying, was good in 4 cases, fairly good in 1 case, and indeterminate in 1 case because of the occurrence of numerous ex-

trasystoles throughout the period of filming.

In the 2 cases in which the examination was carried out by means of injection into the aortic root, aortic insufficiency was not observed. The competence of the mitral valve could be assessed in 5 cases in which

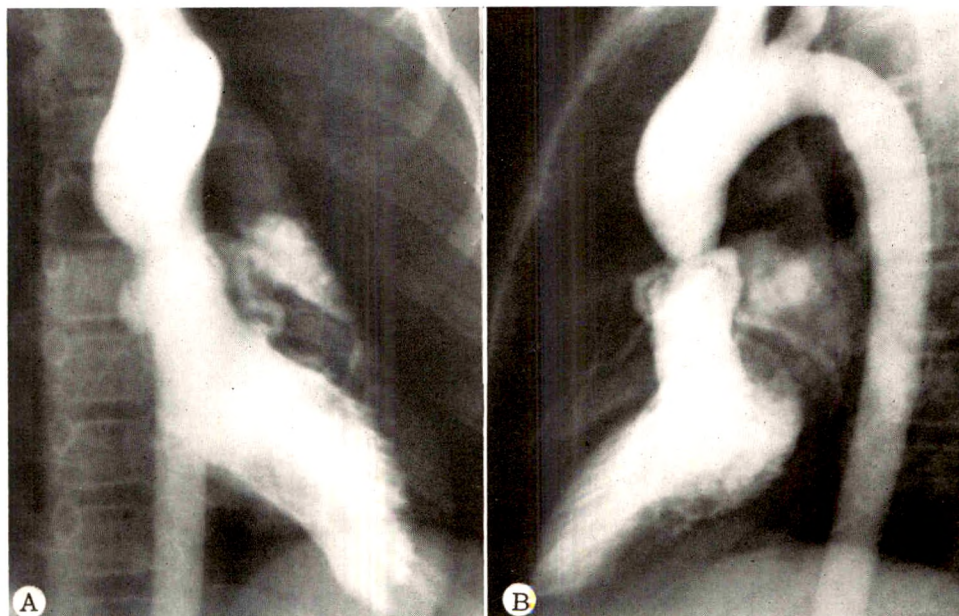


FIG. 13. (A and B) Supravulvular aortic stenosis encircling entire circumference of aortic root in a  $7\frac{1}{2}$  year old girl. Gradient was 98 mm. Hg across stenotic region. Valvular aortic stenosis was also thought to be present, and there was mild mitral insufficiency (note dense opacification of left atrial appendage). This patient also had infantile hypercalcemia.

injections into the left ventricle were carried out, and mitral insufficiency was observed in 1 of these cases.

Evidence of pulmonary stenosis was present in 2 of the 8 cases. One patient had valvular pulmonary stenosis and 1 had infundibular pulmonary stenosis. Peripheral pulmonary-artery stenosis was not evident in any of the cases.

#### SUBVALVULAR AORTIC STENOSIS

*Morphologic Features.* Subvalvular aortic stenosis may be defined as a localized narrowing of the outflow tract of the left ventricle at or below the line of attachment of the aortic cusps. Although many conditions may cause obstruction of the left ventricular outflow tract, if those forms related to left atrioventricular valve abnormalities are excluded, most examples of subaortic stenosis may be regarded as either of the muscular or fibrous type.

Muscular subaortic stenosis classically is seen as a relatively localized bulging of the muscular interventricular septum below the

aortic ring (Fig. 14A). In some cases, similar muscular masses located more on the anterolateral wall of the left ventricle may obstruct the outflow tract. The overlying endocardium is commonly thickened. The bulging muscle may be localized to the basilar portion of the outflow tract, or it may involve the outflow tract diffusely from the base to the apex (Fig. 14B).

The fibrous or membranous type of subaortic stenosis is due to the presence of a fibrous ring, complete or incomplete, in the outflow tract of the left ventricle within 1 to 3 cm. of the aortic valve (Fig. 15, A, B and C).

Although most instances of muscular subvalvular aortic stenosis have been recognized in young adults, this type of lesion also generally is regarded as congenital, and sufficient examples have been encountered in infants and children to support this view.

*Cases Studied.* Of the 9 patients having subvalvular aortic stenosis, 7 had isolated subvalvular stenosis and 2 had combined subvalvular and valvular stenosis. The pa-



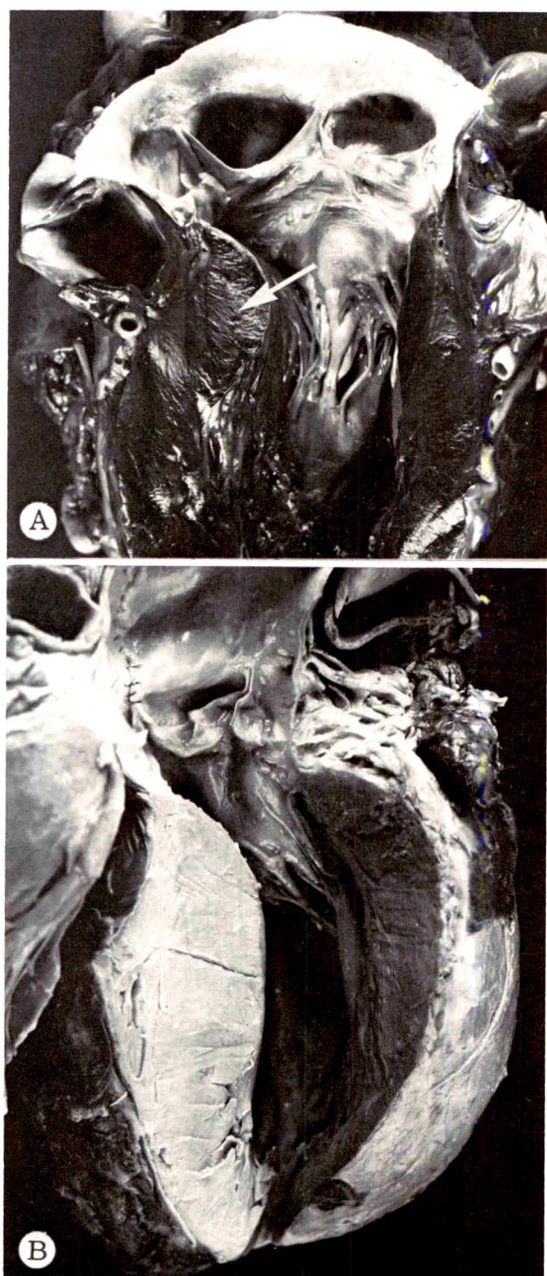


FIG. 14. (A) Opened left ventricle and aortic valve show subvalvular aortic stenosis due to localized muscular hypertrophy, particularly involving ventricular septum (arrow). (B) Opened left ventricle and aortic valve show subvalvular aortic stenosis due to diffuse muscular hypertrophy. Sutures in aortic wall above valve were placed post mortem.

←

slightly enlarged in 2 cases, moderately enlarged in 5 (Fig. 16), and markedly enlarged in 2. The left atrium was believed to be of normal size in 6 cases, slightly enlarged in 2 cases, and moderately enlarged in 1 case (Fig. 17, A and B).

Poststenotic dilatation of the ascending aorta was present in only 1 case of subvalvular aortic stenosis. In that case valvular stenosis was also present.

*Angiocardiographic Findings.* Technique. Left ventriculography, with passage of a catheter in retrograde fashion through the aortic valve, was performed in 8 cases. In 1 case, a left ventriculogram was obtained by means of transthoracic needle puncture of the left ventricle.

Observations. The left ventricular chamber was of normal size in 1 case, slightly smaller than normal in 3 cases, moderately smaller in 2 cases, and markedly smaller in 1 case. In 2 cases, the left ventricular chamber was slightly larger than normal, but in both instances the patients had combined subvalvular and valvular stenosis.

Left ventricular hypertrophy was striking in most of the cases, the thickness of the lateral wall of the left ventricle in diastole ranging from 10 to 40 mm., the average being 17 mm. The hypertrophy of the left ventricular wall was often irregular, resulting in a grossly irregular outline of the left ventricular chamber. Contributing to the irregular outline of the left ventricular chamber in 2 cases was gross hypertrophy of the papillary muscles. The irregular distortion of the left ventricular chamber in most cases of subvalvular aortic stenosis was such that determination of left ventricular

tients, 4 male and 5 female, ranged in age from 7 to 45 years. The average age of those having isolated subaortic stenosis was 29.5 years, and the average age of those having combined subvalvular and valvular stenosis was 8 years.

*Roentgenographic Findings.* The cardiothoracic index ranged from 0.49 to 0.60, the average being 0.54. The left ventricle was



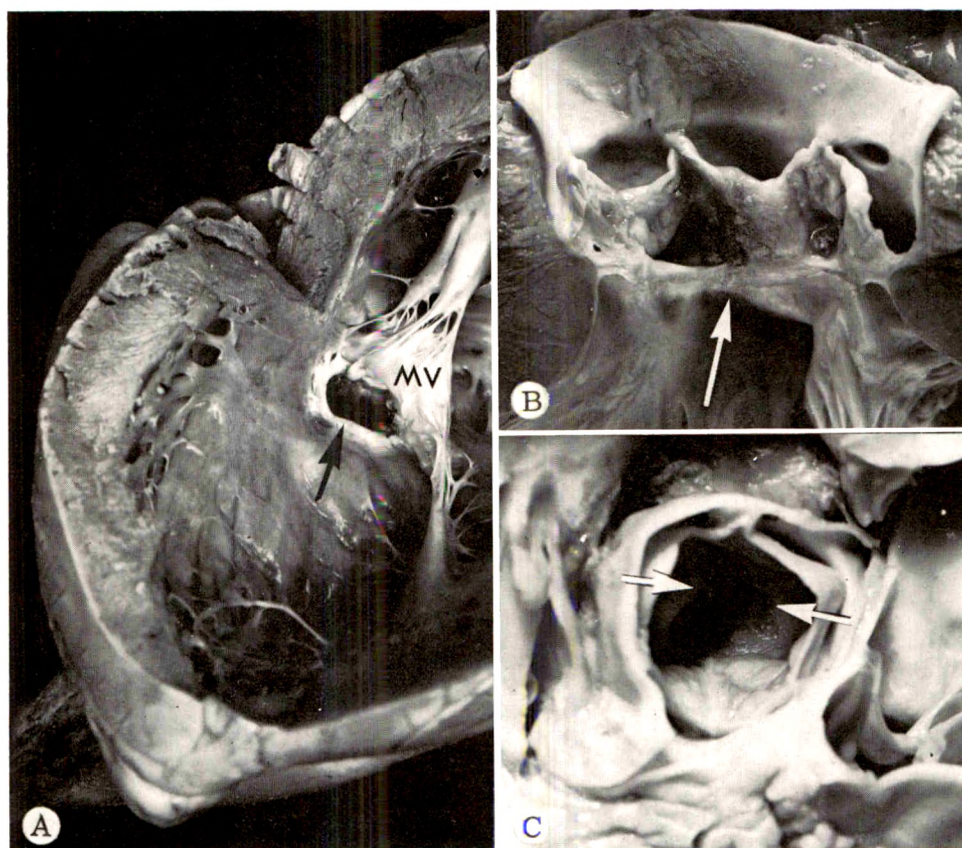


FIG. 15. (A) Subaortic stenosis due to subvalvular fibrous ring or diaphragm (arrow) as viewed from below aortic valve. Mitral valve (MV) is in close proximity to fibrous ring. (B) Subaortic stenosis due to fibrous membrane (arrow). (C) Aortic valve viewed from above with cusps opened to demonstrate subaortic fibrous ring (arrows) producing stenosis.

volume from the angiocardigram was impossible.

The angiocardigram in the usual form of subvalvular aortic stenosis showed evidence of marked septal hypertrophy in the form of a muscular ridge about 2 cm. below the aortic valve (Fig. 18 through 20). This muscular ridge was best visualized in the lateral projection. In ventricular systole the ridge tended to bulge into the outflow tract, producing subvalvular obstruction. The ridge was usually somewhat triangular, with its broad base against the septum. In ventricular diastole, the anterior mitral leaflet closely approximated the hypertrophied septum and the outflow tract assumed the shape of an inverted cone.

The type of left-ventricular outflow-tract obstruction just described was observed in

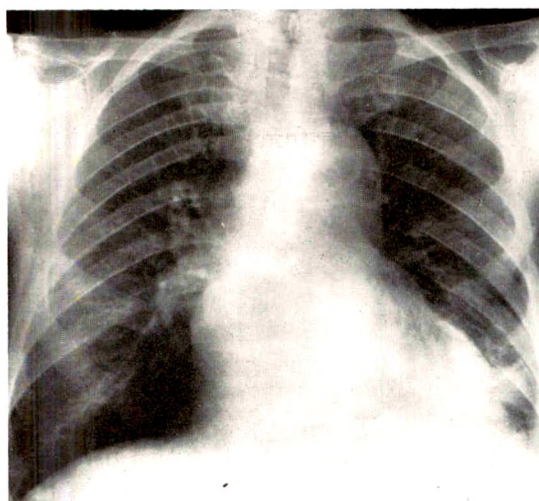


FIG. 16. Subaortic stenosis. Roentgenogram of chest shows moderate enlargement of left ventricle.



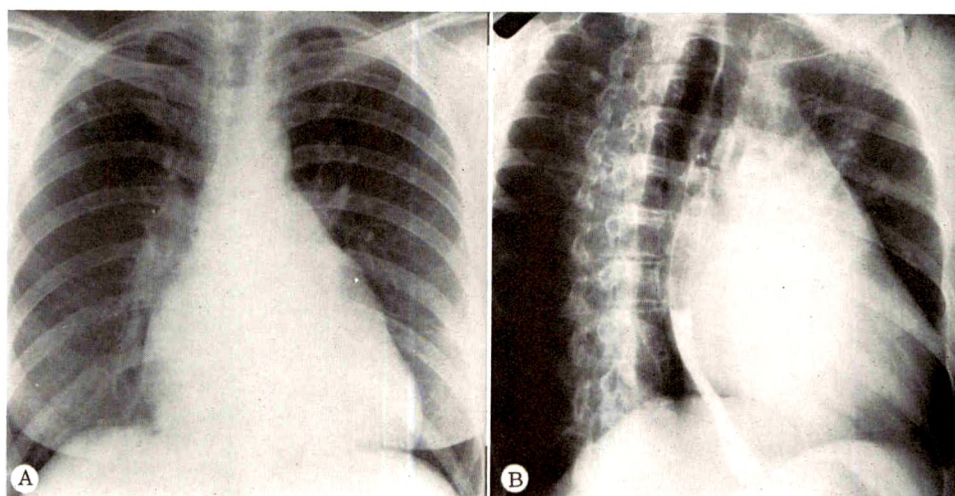


FIG. 17. Subaortic stenosis. (Aortograms of same patient are shown in Figure 18, *A-D*.) (*A*) Roentgenogram of chest shows moderate enlargement of the left ventricle and left atrium. (*B*) Barium swallow study (right anterior oblique projection) shows moderate enlargement of left atrium.

6 of the 9 cases of subvalvular aortic stenosis comprising this series. In 1 case, marked diffuse septal hypertrophy was present and resulted in narrowing of the entire left ventricular outflow tract. In 2 cases, the stenosis, although localized, was relatively low and was located opposite the free margin of the anterior mitral leaflet (Fig. 21, *A-D*; and 22, *A-D*). In 1 patient who exhibited the usual muscular hypertrophy of the septum just below the aortic valve, a second zone of marked localized concentric narrowing was located in the sinus portion of the left ventricle (Fig. 23, *A-D*).

Left ventricular function, as judged from the effectiveness of ventricular contraction and emptying, was good in 5 cases and moderately poor in 2. In 1 case, left ventricular contractions were extremely poor.

The competency of the mitral valve could be assessed in 8 cases in which left ventriculography was carried out. In 4 of these 8 cases, mitral insufficiency was present, the insufficiency being mild in 1 case and moderate in 3 cases.

The aortic valve was tricuspid in all 9 cases. However, in the 2 cases with associated valvular stenosis, the aortic valve

leaflets were thickened and dome-shaped in systole.

Six of the 7 patients having isolated subvalvular aortic stenosis had a normal ascending aorta, without evidence of poststenotic dilatation. One patient with isolated subvalvular stenosis had fairly marked poststenotic dilatation of the ascending aorta. Of the 2 patients having combined subvalvular and valvular stenosis, 1 showed marked poststenotic dilatation and 1 showed slight poststenotic dilatation of the ascending aorta.

A jet of contrast medium extending into the root of the aorta was seen in 2 cases, 1 of isolated subvalvular stenosis and 1 of combined subvalvular and valvular stenosis.

The coronary arteries were well visualized in each instance and were of normal size in 6 cases and of larger size in 3.

#### COMMENT

In isolated congenital valvular aortic stenosis, enlargement of the left ventricle was the most significant single finding on the thoracic roentgenogram. This finding was present in 80 per cent of the cases in our series (Table II). The left ventricular



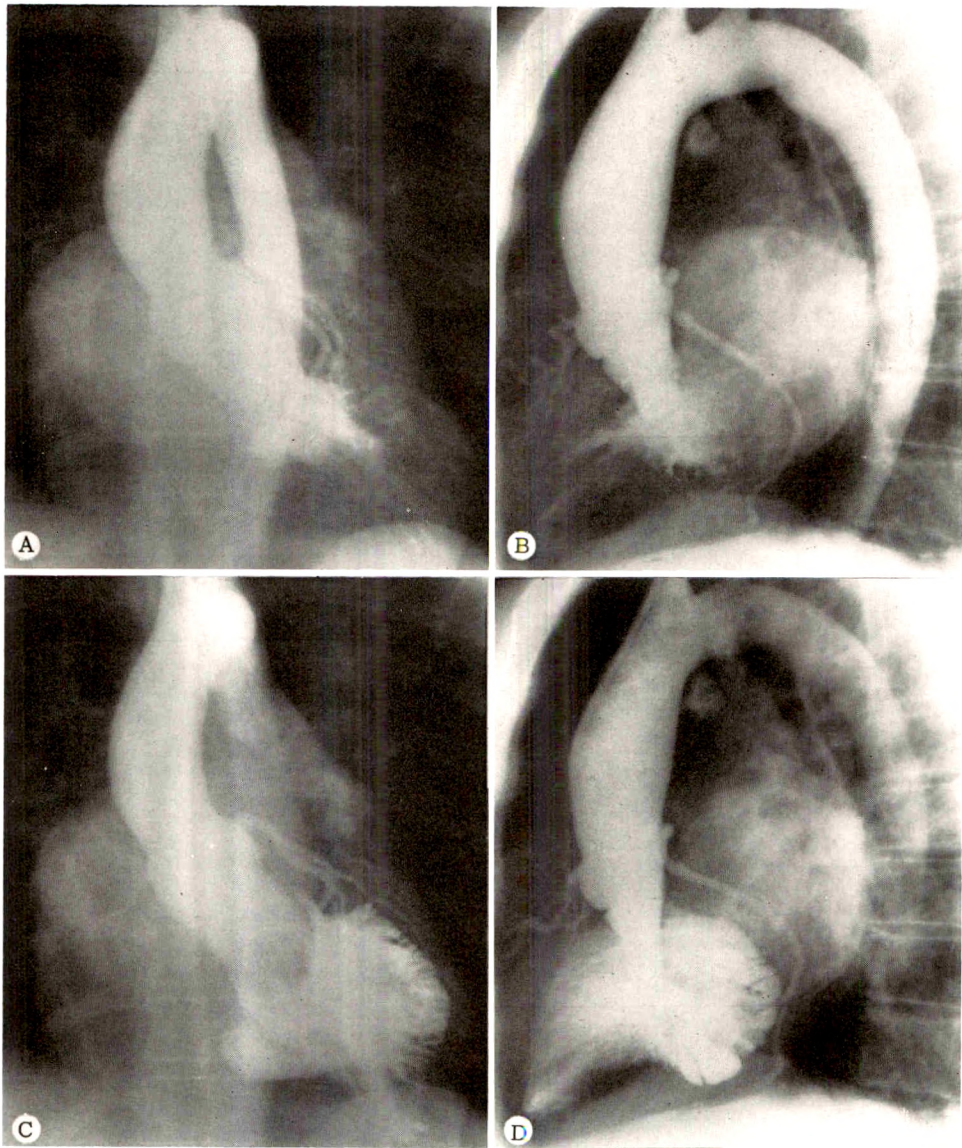


FIG. 18. Subaortic stenosis in a 43 year old woman with pressure gradient of 59 mm. Hg across left ventricular outflow tract. (Roentgenograms of same patient are shown in Figure 17, A and B.) (A and B) Systole, frontal and lateral. (C and D) Diastole, frontal and lateral. Muscular ridge protruding from upper portion of ventricular septum, about 2 cm. below aortic valve, encroaches on outflow tract of left ventricle. Left ventricular outflow tract in diastole shows typical inverted-cone shape (D). There is moderate poststenotic dilatation of ascending aorta. Marked hypertrophy of left ventricular wall and papillary muscles. Mitral insufficiency is evident.

enlargement did not always increase the cardiothoracic index; in some instances it produced only a bulging "left ventricular" contour. This observation is similar to that noted in previous reports<sup>10,20,22</sup> and is in contrast to the findings in acquired valvular

aortic stenosis, in which cases left ventricular enlargement is more pronounced.<sup>19</sup> Left ventricular enlargement was evident in all of the cases of subvalvular aortic stenosis included in the present study. We have found the most marked increases in



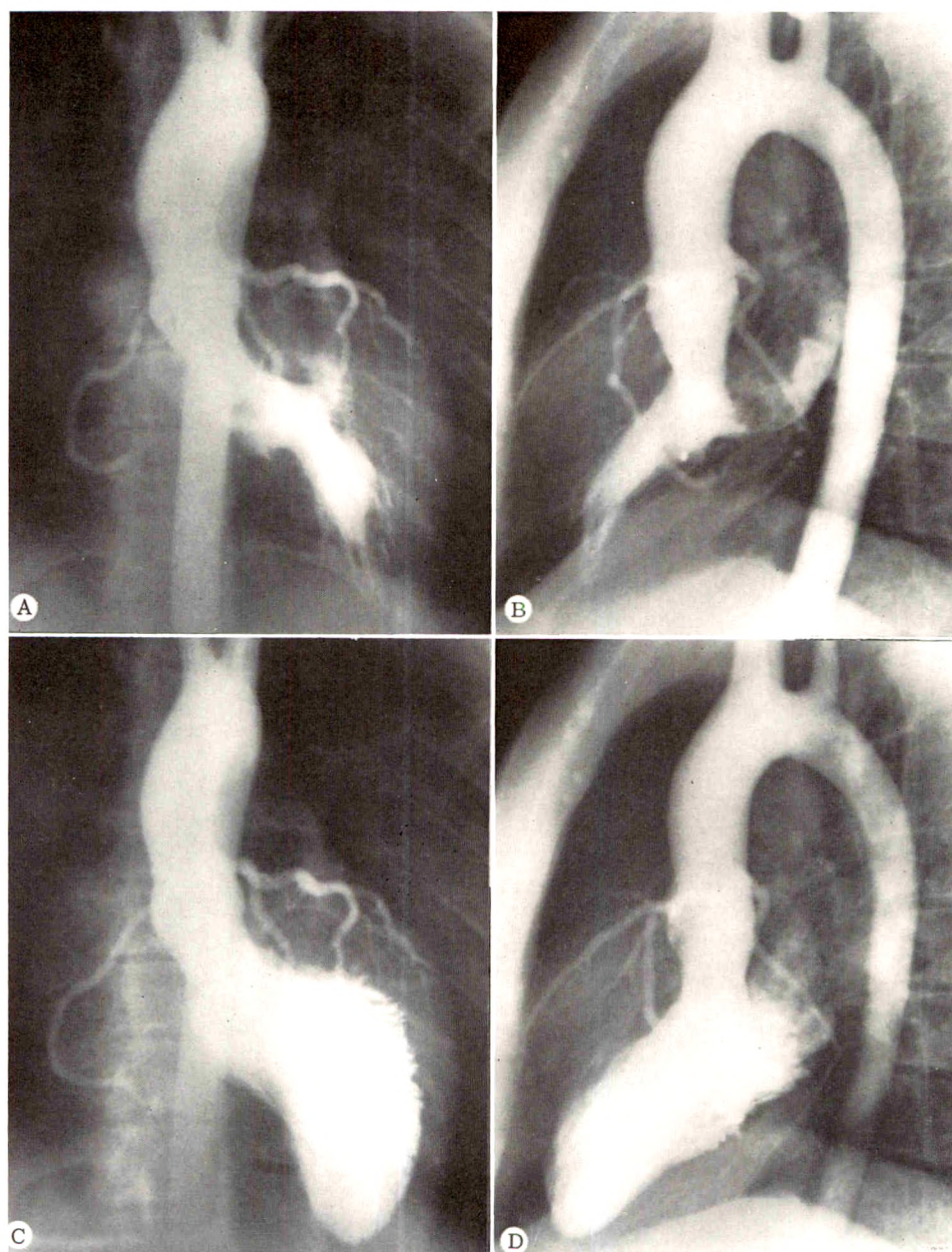


FIG. 19. Subaortic stenosis in a 21 year old woman with gradient of 15 mm. Hg across stenotic region. (A and B) Systole, frontal and lateral. (C and D) Diastole, frontal and lateral. Interventricular septal hypertrophy in usual location, but less pronounced than in patient whose angiocardiograms are shown in Figure 18, A-D. Marked left ventricular hypertrophy.

size of the left ventricle and in the cardiothoracic index in cases of subvalvular aortic stenosis, as have others.<sup>9,10,22</sup> Like Klatte and his co-workers,<sup>22</sup> we found no correlation between the cardiothoracic index and the systolic pressure gradient in any group. In contrast to cases of valvular or sub-

valvular aortic stenosis, left ventricular enlargement was present in only 25 per cent of cases of supra-ventricular stenosis, and in most of these cases the enlargement was slight.

Poststenotic dilatation of the ascending aorta as noted on thoracic roentgenograms



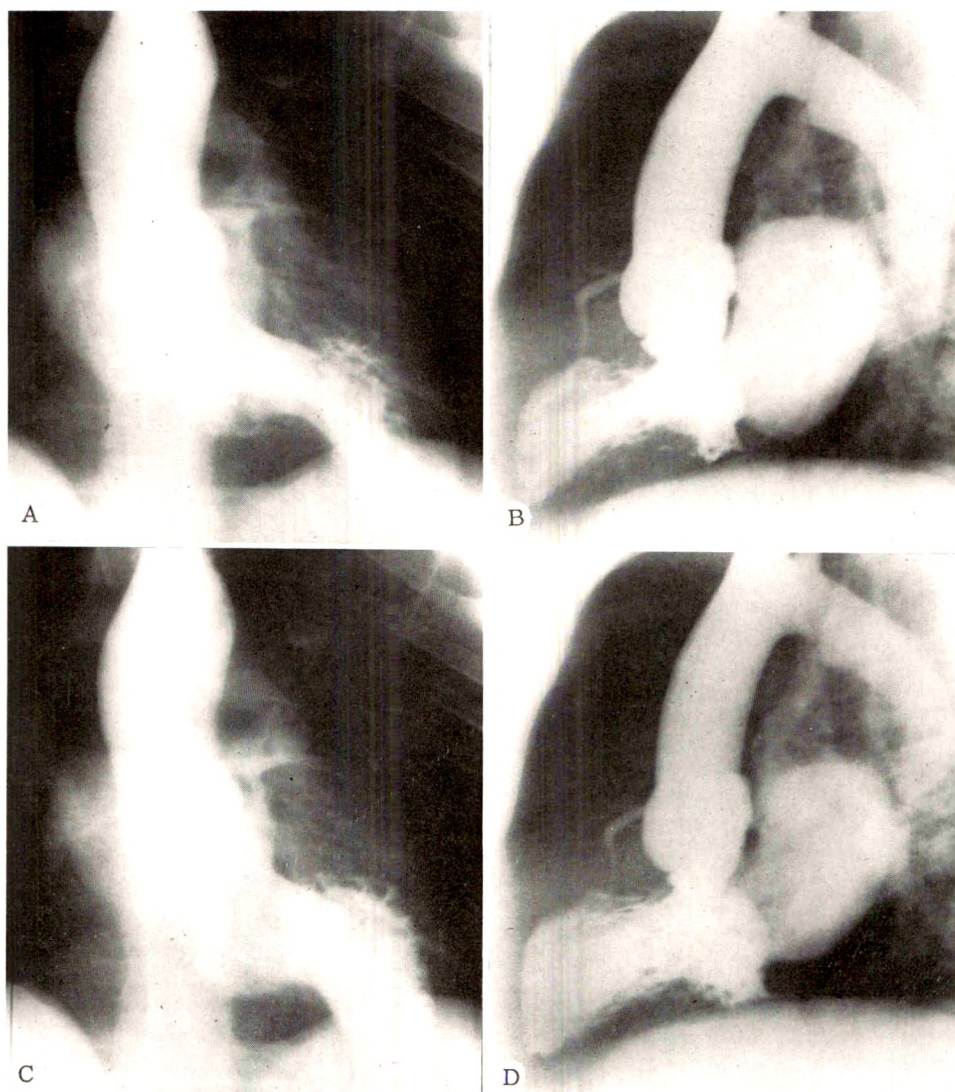


FIG. 20. Subaortic stenosis in a 30 year old man; usual location and with gradient of 16 mm. Hg across stenotic region. (A and B) Systole. (C and D) Diastole. Marked irregular hypertrophy of left ventricle. Moderate mitral insufficiency.

was present in 35 per cent of cases with valvular aortic stenosis, which is in keeping with the findings of Straube and co-workers,<sup>41</sup> but which represents only half the frequency of poststenotic dilatation observed by Braunwald and co-workers<sup>10</sup> and Klatte and co-workers.<sup>22</sup> In contrast, poststenotic dilatation of the aorta was not observed in the plain roentgenograms of the chest of patients having supra- or subvalvular aortic stenosis, with the exception of 1 case of combined subvalvular and valvular stenosis. Although Klatte and co-

workers<sup>22</sup> found poststenotic dilatation of the aorta in 71 per cent of cases of subvalvular aortic stenosis and stated that poststenotic dilatation of the aorta was not a good differential point between congenital valvular and subvalvular types of aortic stenosis, our studies would indicate that the presence or absence of poststenotic dilatation of the aorta may be of some help in this differential diagnosis.

Left atrial enlargement, in our series, was found in 2 (8.5 per cent) of 23 cases of isolated valvular aortic stenosis and in 3



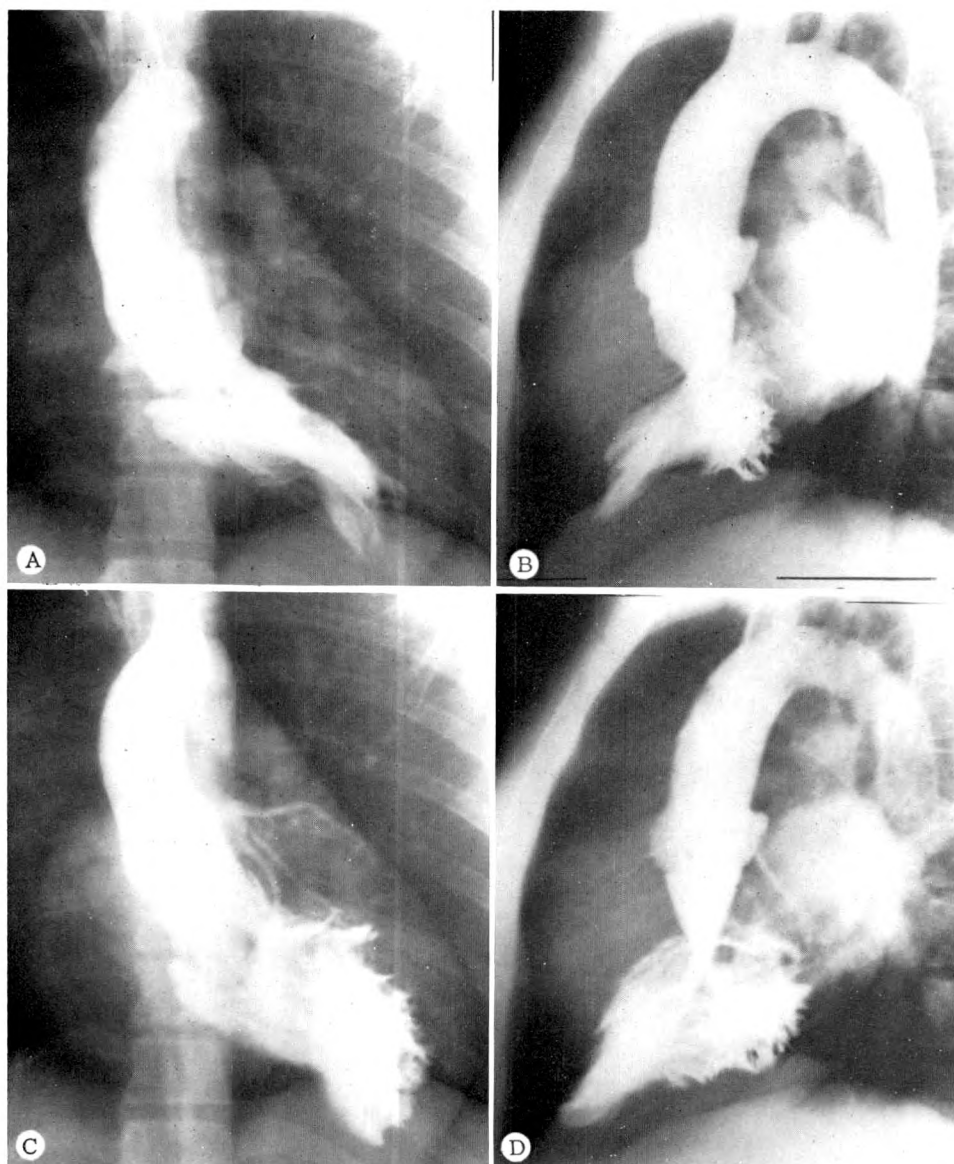


FIG. 21. Angiocardio gram of a 26 year old woman with subaortic stenosis and gradient of 94 mm. Hg across stenotic region. (*A* and *B*) Systole. (*C* and *D*) Diastole. Hypertrophy of the interventricular septum is located slightly lower than usual and is opposite free margin of anterior mitral leaflet. Note inverted-cone appearance of left ventricular outflow tract in diastole. (*D*) Note hypertrophic ridge is also well visualized on anteroposterior angiocardio gram. Mitral insufficiency is present.

(33 per cent) of 9 cases of subvalvular aortic stenosis. Klatte and co-workers<sup>22</sup> reported frequencies of 35 per cent and 43 per cent, respectively for these two groups, while Braunwald and co-workers<sup>10</sup> observed left atrial enlargement in 9 per cent of cases of aortic stenosis, including all types. No enlargement of the left atrium was found in cases of supra valvular stenosis

in our series or in those cases which have been reported in the literature.

Associated lesions such as coarctation of the aorta, patent ductus arteriosus, and ventricular septal defect have been reported in previous studies of aortic stenosis.<sup>1,7,10,12,14,16,18,21,26,28,29,37-39</sup> In our series, cases of coarctation of the aorta and patent ductus arteriosus were found in association

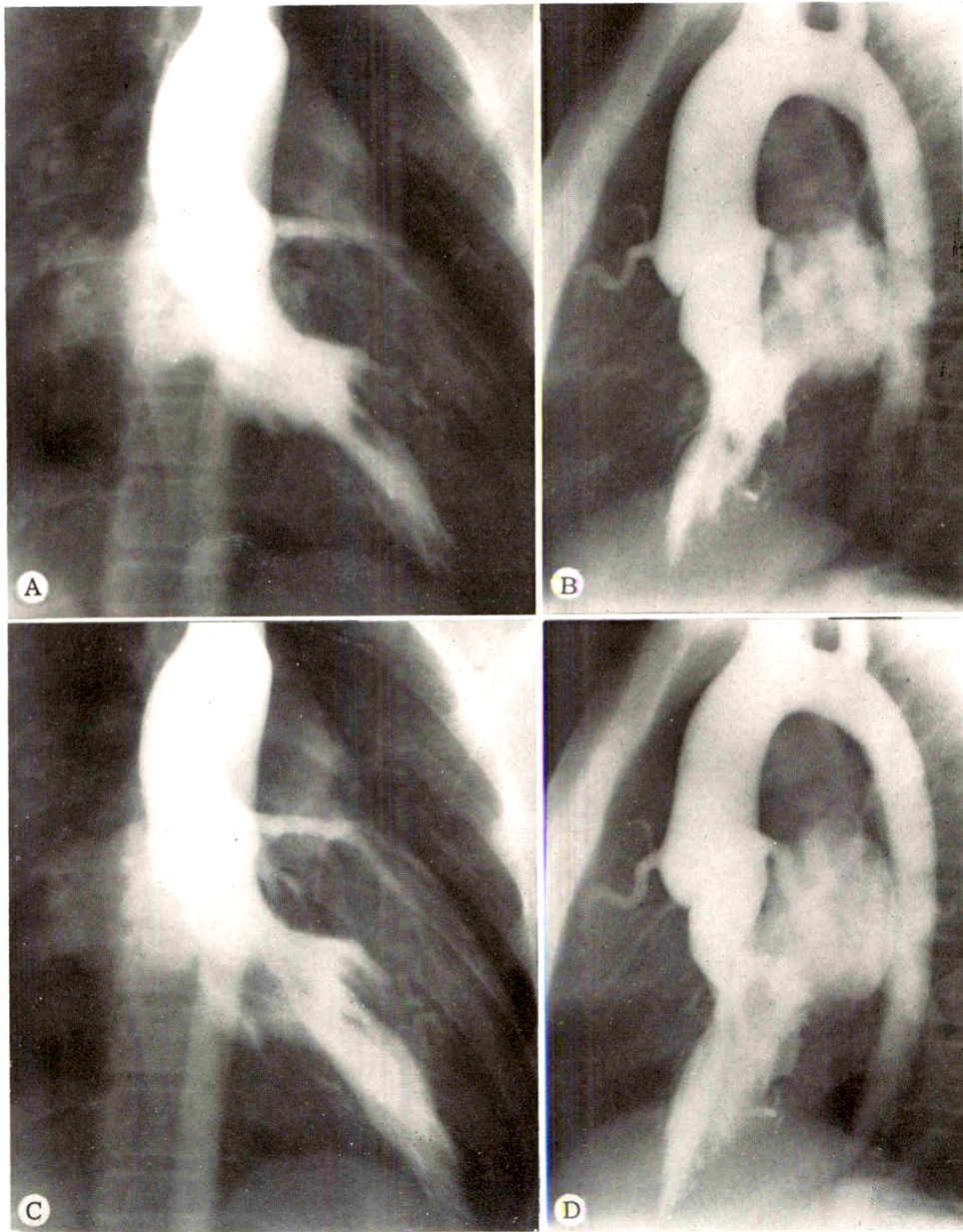


FIG. 22. Subaortic stenosis in a 13 year old girl. Gradient was 70 mm. Hg across stenotic region. (A and B) Systole. (C and D) Diastole. Hypertrophic muscular ridge is visible opposite free margin of anterior mitral leaflet. Location is similar to that in case illustrated in Figure 21, A-D. Marked left ventricular hypertrophy. Mitral insufficiency is present.

with valvular aortic stenosis only. A relatively high incidence of peripheral pulmonary-artery stenosis in association with supravalvular aortic stenosis has been reported by Bourassa and Campeau<sup>8</sup> and by Watson.<sup>43</sup> No cases of peripheral pulmonary artery stenosis were detected in our group of patients with supravalvular stenosis,

although 2 patients had pulmonary stenosis. In 1 of these, there was associated valvular pulmonary stenosis, and in another, there was associated infundibular pulmonary stenosis.

Calcification of the aortic valve was not found in any of our cases of valvular aortic stenosis, although such calcification has



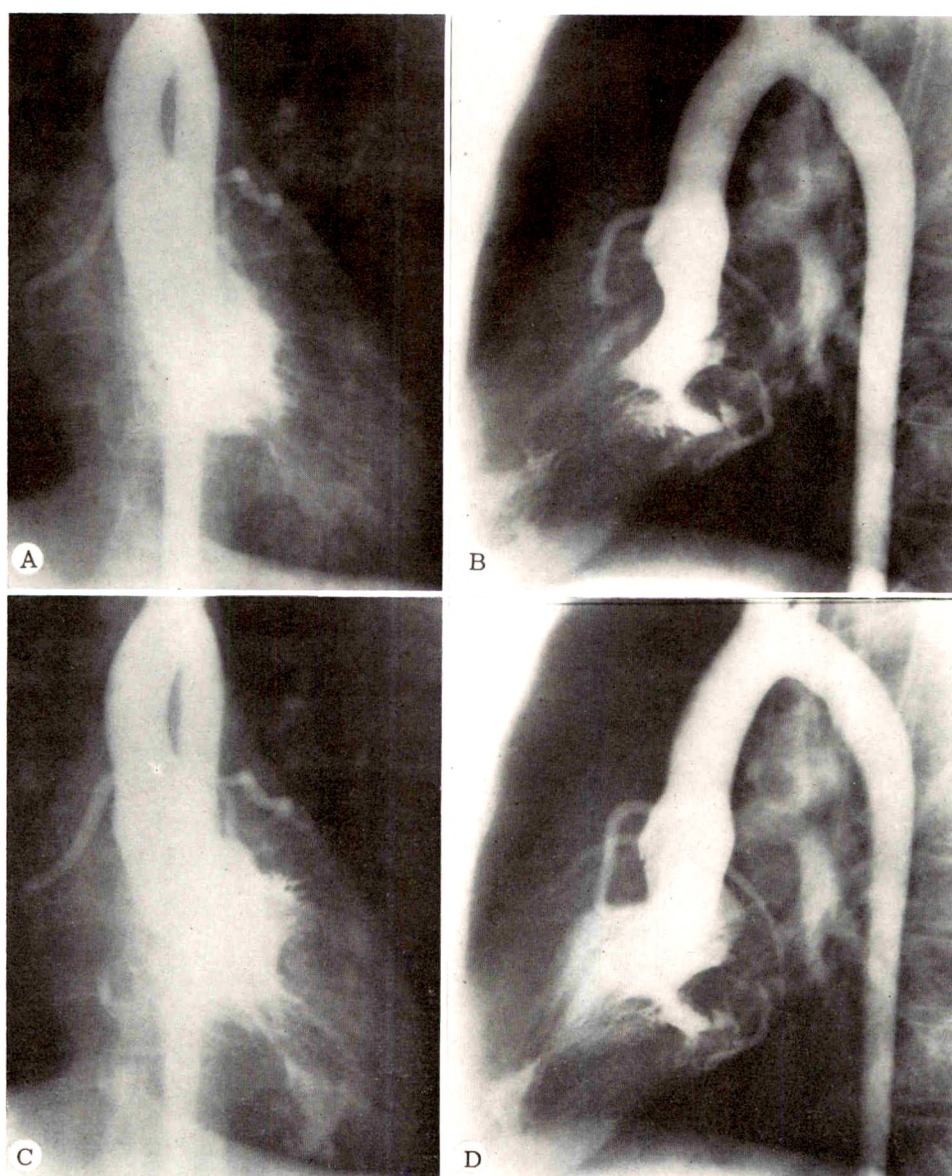


FIG. 23. Angiocardio gram of a 29 year old man. (A and B) Systole. (C and D) Diastole. There is subaortic stenosis in usual location, about 2 cm. below aortic valve and opposite midportion of anterior mitral leaflet. In addition, there is extreme hypertrophy of left ventricular myocardium with second zone of obstruction or stenosis in sinus portion of left ventricle well below mitral valve. Two gradients were present—one of 10 mm. Hg across the subvalvular zone, and one of 35 to 40 mm. Hg across the stenotic zone in sinus portion of ventricle. As in most cases of subaortic stenosis, irregular contour of left ventricular chamber made left ventricular volume measurements impossible.

been reported by Braunwald and co-workers,<sup>10</sup> who found calcification in 18 per cent of such cases.

The angiocardio graphic findings in congenital aortic stenosis have been described by various authors.<sup>5, 9, 11, 15, 21, 24, 27, 30-32, 40</sup> As

summarized by Björk, Cullhed, and Lodin,<sup>5</sup> the angiocardio graphic diagnosis of valvular aortic stenosis is based on (1) thickening of the aortic cusps, (2) rigidity of the cusps, (3) small caliber of the aortic orifice, (4) slow rate of flow of contrast medium

TABLE II  
MAJOR ROENTGENOGRAPHIC SIGNS IN CONGENITAL  
AORTIC STENOSIS: 40 CASES

Type of Stenosis	Roentgenologic Findings (percent)		
	Enlarge- ment of Left Ventricle	Post- stenotic Dilatation of Aorta	Enlarge- ment of Left Atrium
Valvular	80	35	8.5
Supravalvular	25	0	0
Subvalvular	100	0	33

through the orifice, (5) dilatation of the ascending aorta, (6) slow emptying capacity of the left ventricle, (7) thickening of the left ventricular wall, and (8) dome-shaped aortic valve. In addition, the orifice of the aortic valve in congenital valvular aortic stenosis is eccentric in many cases and tends to be smaller in the patients with the largest gradients across the stenosis. These various points were confirmed by this present study.

The angiocardiographic findings in supravalvular stenosis as described by several authors<sup>11,15,17,25,30,32,40</sup> were confirmed in this study. The main finding was the presence of a short segment of narrowing of the aortic root at or just above the point of insertion of the aortic leaflets. One of our patients exhibited the syndrome consisting of unusual facial features and mental retardation in association with supravalvular aortic stenosis, as has been described in the literature.<sup>3,4,33,44,45</sup> One patient of this group had infantile hypercalcemia, as was noted in 1 of the group of cases reported by Black and Bonham Carter.<sup>6</sup>

The etiology and nature of subvalvular aortic stenosis have been much debated in the recent literature. Although the concept that muscular subaortic stenosis may be an acquired abnormality in some cases<sup>42</sup>—perhaps occurring secondary to idiopathic myocardial hypertrophy—has been advanced, its occurrence in children and in some adults without evidence of marked diffuse myocardial disease suggests that the

entity may be in many cases a congenital lesion. The diagnosis of this condition and its distinction from other conditions producing left ventricular outflow obstruction can be made accurately by means of angiocardiology. Although the association of subvalvular pulmonary stenosis with subvalvular aortic stenosis has been occasionally observed,<sup>2,23,34–36,38</sup> no such association was found in our series.

SUMMARY

The roentgenographic and angiocardiology features of congenital valvular, supravalvular, and subvalvular aortic stenosis have been reviewed and described.

The single most common roentgenographic finding in this group of conditions was left ventricular enlargement. Poststenotic dilatation of the ascending aorta was frequently discernible on the roentgenograms of the chest of patients having congenital valvular stenosis, but it was not detected in patients having isolated supravalvular or subvalvular stenosis. Left atrial enlargement was common in subvalvular stenosis, was infrequently encountered in valvular stenosis, and was not detectable in any cases of supravalvular stenosis.

The pathologic anatomy of the various types of congenital aortic stenosis was well demonstrated in all instances by means of large-film biplane selective angiocardiology.

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## SUPRAVALVULAR AORTIC STENOSIS\*

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**S**UPRAVALVULAR aortic stenosis may occur as an isolated entity, but its frequent association with peripheral pulmonary artery stenosis, physical and mental retardation and a characteristic abnormal facies with maldeveloped dentition has been emphasized recently.<sup>8</sup> These findings comprise one or more new and fascinating syndromes. In addition, recent studies<sup>9,30</sup> indicate that hypercalcemia in infancy may be an additional facet of the syndrome and may actually be responsible for the various developmental abnormalities.

As a cause of obstruction to left ventricular outflow which is clearly operable, its recognition and differentiation from valvular or subvalvular stenosis are important prior to surgery. The present report is designed to emphasize certain roentgenologic features which have not yet received adequate attention, as well as to relate the anatomic lesion to the total clinical picture.

## REPORT OF SPECIFIC CASES

**CASE I.** S. R., a 12 year old white boy, had a heart murmur first noted at the age of 1 month. He had been entirely well until 3 months before admission, when he first complained of dyspnea on exertion and occasional sharp chest pain along the left sternal margin. He had two bouts of syncope following severe exertion, with subsequent restriction of his activities.

Physical examination revealed a height of 4 feet, 8 inches (10th percentile) and a weight of 92 pounds (40th percentile). The facies was normal. Blood pressure was 130/60. The pulses were normal and equal. Visible pulsation was present in the suprasternal notch and along the carotids with a systolic thrill palpable over the carotids. A Grade 4 systolic murmur was noted in the aortic area with transmission to the neck and apex. The aortic second sound was normal.

The remainder of the examination was normal. An electroencephalogram showed left ventricular hypertrophy.

Roentgenograms of the chest (Fig. 1, *A* and *B*) showed only minimal left ventricular prominence. The aorta was not significantly dilated.

Cardiac catheterization was performed via a left ventricular puncture. The left ventricular pressure was 176/0, the aortic pressure 130/84. Several withdrawal pressure recordings were obtained, demonstrating an area of supravascular stenosis with a systolic gradient of 46 mm. of mercury.

A retrograde aortogram (Fig. 1*C*) and cine-aortography showed a definite localized area of supravascular aortic stenosis with dilated aortic sinuses and prominent coronary arteries. The aortic leaflets were normal. There was slight but definite narrowing of the origin of the left common carotid artery.

At surgery, there was a localized segmental narrowing at the level of the attachments of the commissures to the aortic wall. The aorta was incised longitudinally and a fibromuscular band was noted around the entire circumference of the aorta at the level of the attachments of the commissures. The incision was prolonged into the noncoronary sinus and the aortic lumen widened. A teardrop-shaped woven teflon patch was inserted.

The patient did well after surgery with an increased exercise tolerance and a decrease of the murmur to Grade 1 when last seen, 16 months after surgery.

**CASE II.** M.V., a 15 year old white boy, complained of fainting and dizzy spells lasting up to 1 minute in duration over the prior 6 months. One fainting episode occurred after exercise but the over-all exercise tolerance had been excellent. There was no history of chest pain, dyspnea or previous heart disease.

Physical examination revealed a blood pressure of 145/60 in the right and 110/80 in the

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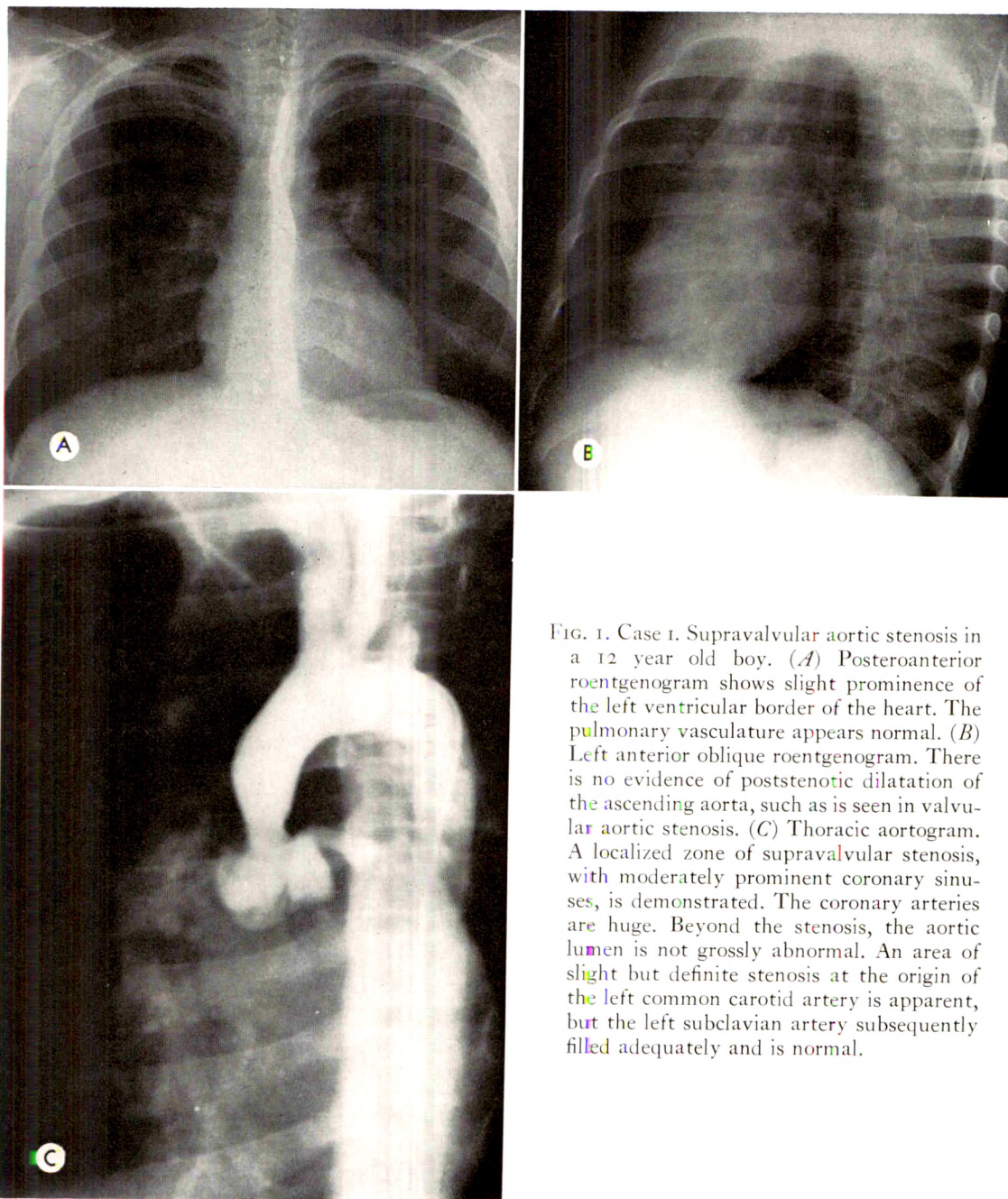


FIG. 1. Case 1. Supravulvular aortic stenosis in a 12 year old boy. (A) Posteroanterior roentgenogram shows slight prominence of the left ventricular border of the heart. The pulmonary vasculature appears normal. (B) Left anterior oblique roentgenogram. There is no evidence of poststenotic dilatation of the ascending aorta, such as is seen in valvular aortic stenosis. (C) Thoracic aortogram. A localized zone of supravalvular stenosis, with moderately prominent coronary sinuses, is demonstrated. The coronary arteries are huge. Beyond the stenosis, the aortic lumen is not grossly abnormal. An area of slight but definite stenosis at the origin of the left common carotid artery is apparent, but the left subclavian artery subsequently filled adequately and is normal.

left arm. The right carotid artery was palpably stronger than the left and a thrill was noted on the left. The facies was normal. There was no clinical evidence of cardiomegaly, but a rough Grade 5 of 6 systolic murmur was heard over the entire precordium with radiation to the back. The remainder of the physical examination was normal.

The electrocardiogram was normal.

Roentgenograms of the chest (Fig. 2, A and B) showed no evidence of cardiac enlargement or poststenotic dilatation of the aorta.

Cardiac catheterization demonstrated an abrupt systolic gradient at the supravalvular level with an aortic root pressure of 200/98 and a pressure in the aortic arch of 142/98, yielding



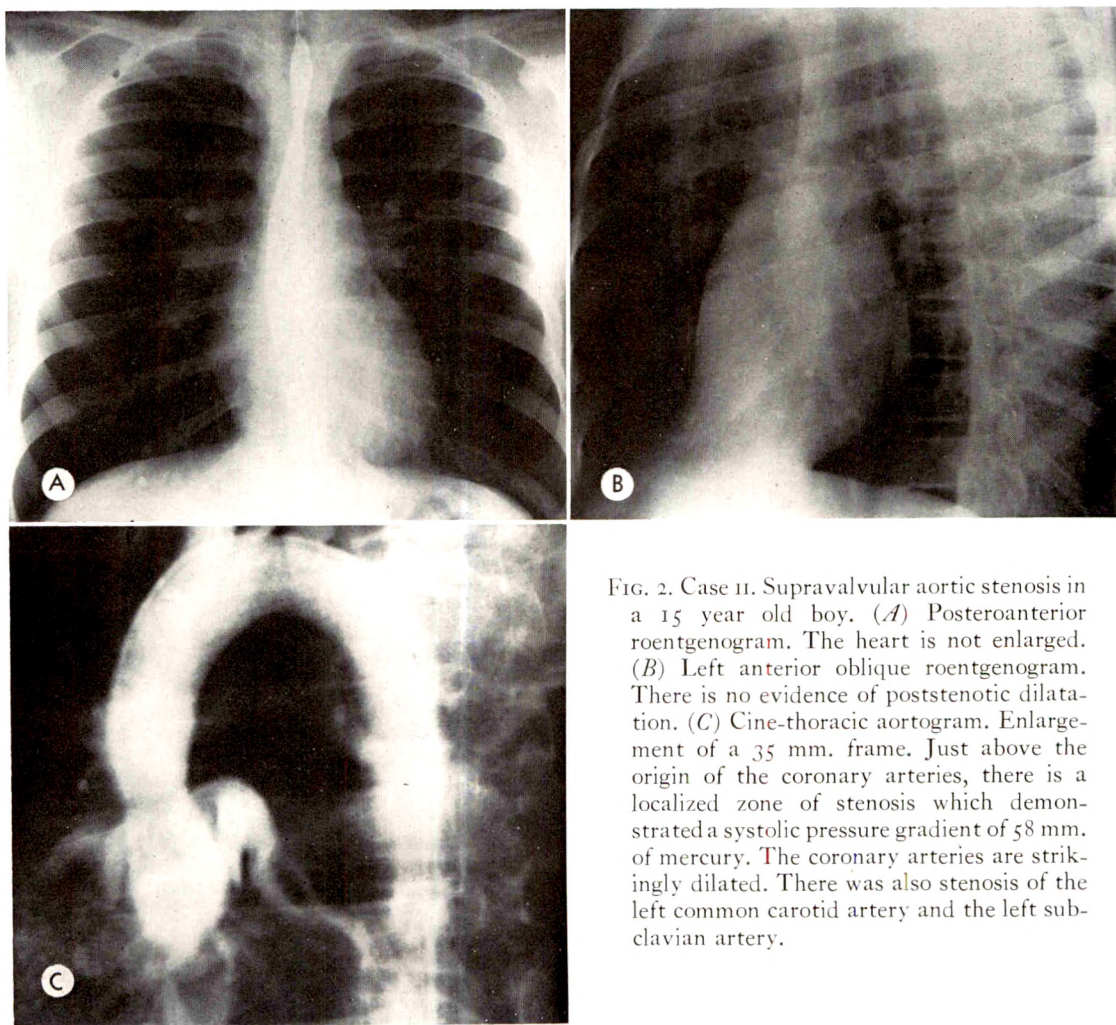


FIG. 2. Case II. Supravulvar aortic stenosis in a 15 year old boy. (A) Posteroanterior roentgenogram. The heart is not enlarged. (B) Left anterior oblique roentgenogram. There is no evidence of poststenotic dilatation. (C) Cine-thoracic aortogram. Enlargement of a 35 mm. frame. Just above the origin of the coronary arteries, there is a localized zone of stenosis which demonstrated a systolic pressure gradient of 58 mm. of mercury. The coronary arteries are strikingly dilated. There was also stenosis of the left common carotid artery and the left subclavian artery.

a mean systolic pressure gradient of 58 mm. of mercury.

A retrograde thoracic aortogram (Fig. 2C) showed discrete narrowing of the aorta approximately 3 cm. above the sinuses with an aortic lumen of approximately 1.5 cm. at this level. There was stenosis at the origin of the left common carotid artery. The coronary arteries were dilated and tortuous. There was no evidence of aortic valvular stenosis or insufficiency.

At surgery, a 4 cm. right aortotomy was performed. Marked hypertrophic supravulvar stenosis was identified with an internal diameter in the aortic lumen of about 1.0 cm. The aortic intima was markedly thickened. The incision was widened with a teardrop-shaped teflon patch. The patient tolerated this procedure well and was discharged on his ninth postoperative day to be followed in clinic. His subsequent course was uneventful.

CASE III. A.H., an 11 year old white boy, was born with respiratory distress and poor muscle tone. A diagnosis of mongolism with heart disease was made during his first year. His growth and development were slow and the IQ was in the 75-80 range at age 4 years. His chief complaint was fatigue with excessive activity. No other cardiac symptoms were noted. He was described as an unusually affable and cooperative child.

Physical examination revealed a height in the 40th percentile and a weight in the 25th percentile. The blood pressure was 90/60 in the right arm and 80/60 in the left arm and 80/70 in the left leg. The patient had an unusual face with large low set ears but no hypertelorism or micrognathia. He was friendly and talkative during examinations. The left brachial pulse was weaker than the right and the femoral pulsations were also slightly weakened. There



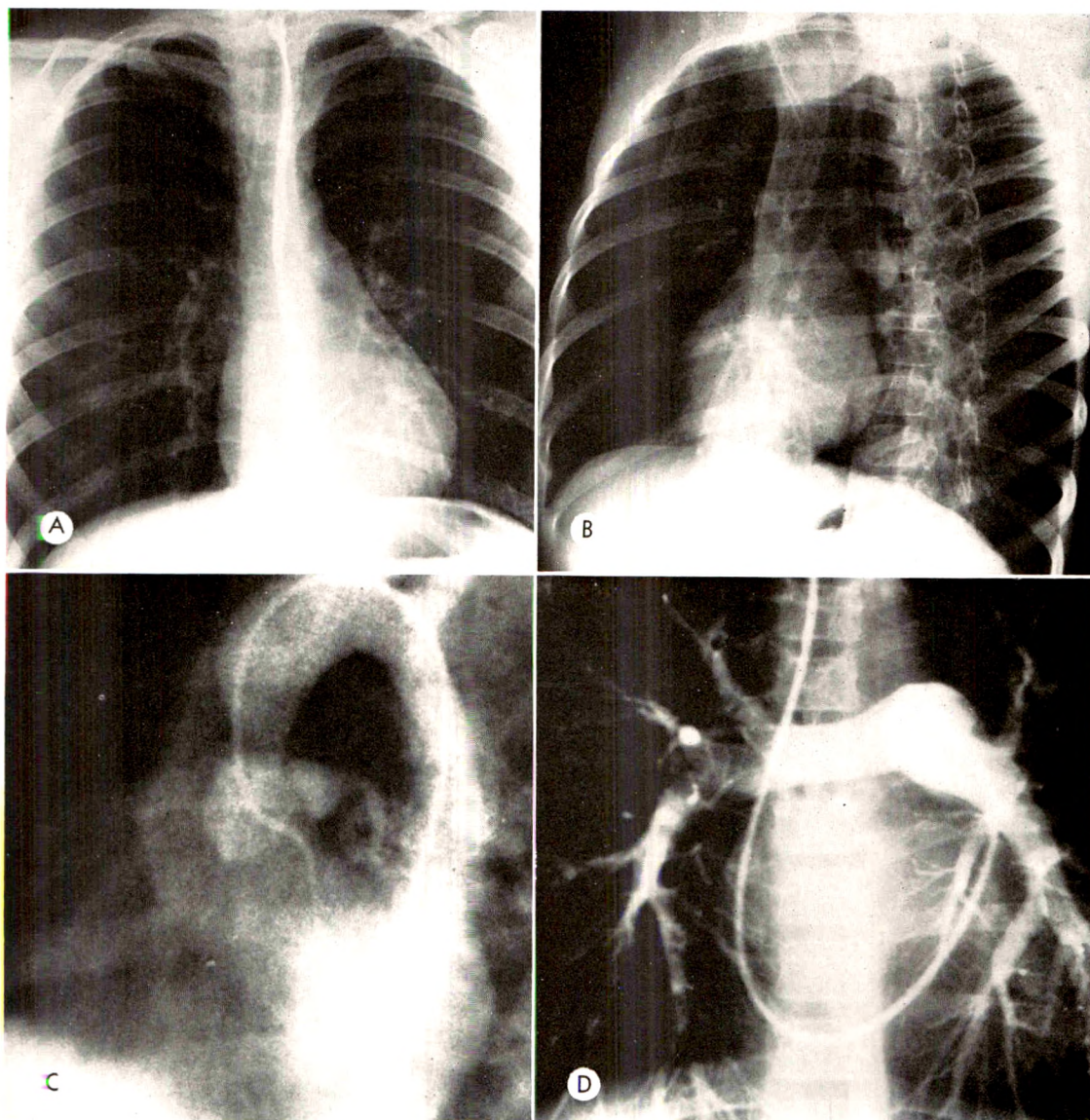


FIG. 3. Case III. Supravalvular aortic stenosis in an 11 year old boy. (A) Posteroanterior roentgenogram. No evidence of significant cardiac enlargement is visible. (B) Left anterior oblique roentgenogram. The left ventricle projects slightly posteriorly, but is clearly not significantly dilated. No evidence of poststenotic dilatation of the aorta is visible. (C) Cine-thoracic aortogram. Enlargement of a 35 mm. frame. A localized area of narrowing is visible just above the aortic sinuses. The coronary arteries are dilated and tortuous. Beyond the stenosed area the aorta gradually widens into the isthmus. The origins of the brachiocephalic vessels are normal. A peak systolic pressure gradient of 80 mm. mercury is found. (D) Selective pulmonary arteriogram. There is no evidence of branch stenoses.

was clinical evidence of left ventricular enlargement with a thrill in the suprasternal notch. A Grade 4 of 6 long, harsh systolic ejection murmur was noted in the first intercostal space near the sternum. There was no ejection click.

Electrocardiograms showed left ventricular

predominance and hypertrophy. The serum calcium was 9.4 mg. per cent.

Roentgenograms of the chest (Fig. 3, A and B) showed no abnormalities.

Cardiac catheterization revealed a left ventricular pressure of 175/6, a supravalvular aortic pressure of 167/58, and ascending aortic



pressure of 95/66. The peak systolic pressure gradient was 80 mm. of mercury. The right ventricular pressure was 21/3, the main pulmonary pressure 20/6, and the right pulmonary artery pressure 17/6.

Cine-thoracic aortography delineated a localized area of narrowing just above the sinuses of Valsalva with a modest degree of poststenotic dilatation beyond (Fig. 3C). The aortic valve leaflets were normal, as were the origins of the brachiocephalic vessels. The coronary arteries were dilated and tortuous. A pulmonary arteriogram was normal (Fig. 3D).

At surgery, a localized stenosis was noted at the base of the aorta with dilatation of the aorta at the level of the cusps. A longitudinal 5 cm. aortotomy was made into the noncoronary sinus which revealed a slightly thickened aortic valve with slight underdevelopment of the right cusp. At the area of stenosis, the diameter of the aortic lumen was approximately 4 mm. and both internal and external constrictions were present. A repair was completed by insertion of a teflon patch. The patient did well post surgery and was discharged on his tenth postoperative day. He continued to be asymptomatic, as observed in the cardiac clinic 1 month after surgery.

CASE IV. J.W., a 9 year old white male, had a heart murmur, first heard at the age of 6 months. Since that time occasional bouts of cyanosis of the lips and fingernails, shortness of breath, and a limited exercise tolerance with leg pains had been noted. A clinical diagnosis of aortic stenosis was made at the age of 5 years. At 7½ years, glomerulonephritis and rheumatic fever required a period of bed rest lasting 6 months.

Physical examination revealed a blood pressure of 120/100 in the right arm, 100/80 in the left arm, and 120/80 in the left leg. His height was in the 30th percentile and his weight in the 80th percentile. A weak radial pulse was noted on the left. There was no clinical evidence of cardiomegaly, but a Grade 2 of 6 systolic thrill was palpable at the second intercostal space. A Grade 4 of 6 harsh systolic murmur was heard in the second intercostal space along the right sternal border with radiation over the entire precordium and to the neck and back. There was no ejection click. The remainder of the physical examination was entirely normal.

An electrocardiogram revealed left ventricular hypertrophy. Serum calcium was 10.9 mg. per cent.

Roentgenograms of the chest showed only slight prominence of the left ventricular border (Fig. 4, A and B).

At cardiac catheterization, the left ventricular pressure was 189/3, the proximal ascending aortic pressure 190/54, and the distal ascending aortic pressure was 73/54 with a mean systolic pressure gradient of 87 mm. of mercury. Pressure in the right ventricle was 32/3, pressure in the main pulmonary artery 38/4, pressure in the left pulmonary artery 21/7 and pressure in the right pulmonary artery 12/3.

Cine-thoracic aortography revealed severe supravalvular aortic stenosis for several centimeters above the sinus of Valsalva (Fig. 4C). There was some hypoplasia of the entire ascending aorta. Marked dilatation and tortuosity of the coronary arteries were noted, particularly on the right. The aortic valve leaflets were normal. Stenosis was present at the origin of the left common carotid artery and, to a lesser extent, at the origin of the innominate artery with tortuosity and poststenotic dilatation. A pulmonary arteriogram showed stenosis of the proximal portion of the left main pulmonary artery, at the origin of the right pulmonary artery, and in multiple branch pulmonary vessels (Fig. 4 D). Surgical correction has been recommended but has not yet been undertaken.

## DISCUSSION

### I. ETIOLOGY AND PATHOGENESIS

Supravalvular aortic stenosis is a lesion of obscure congenital origin. Beuren *et al.*,<sup>8</sup> impressed by the associated pulmonary branch stenoses, suggested that the dual involvement indicated a defective formation of the aortic septum as the etiologic factor. Such a concept is not tenable because the major pulmonary involvement is in the branches of the pulmonary artery, arising from the sixth brachial arches, rather than the aortic septum. Whatever the defect is, it clearly involves many tissues, including blood vessels, brain, teeth and bone.

Eisenberg *et al.*<sup>25</sup> have studied the familial aspects of the anomaly. Three generations in 2 families comprising almost 100 persons were investigated for the presence of supravalvular aortic stenosis. There were 5 proven cases, 8 cases in which the diagnosis was tentatively made by the char-

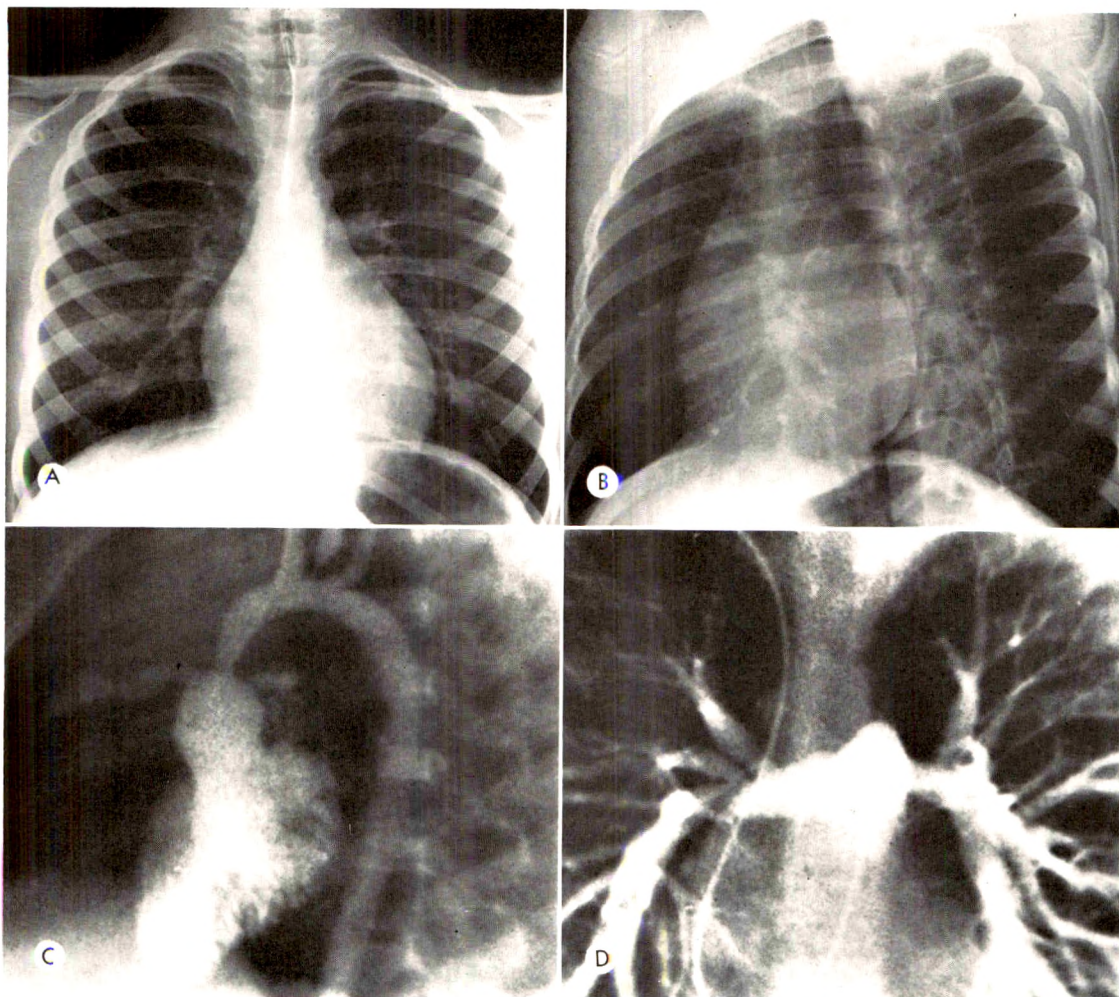


FIG. 4. Case IV. (A) Posteroanterior roentgenogram. The heart is at the upper limits of normal size, but with some fullness of the lower left heart border. (B) Left anterior oblique roentgenogram. Moderate prominence of both left and right ventricles is apparent. No evidence of poststenotic dilatation of the aorta is seen. (C) Cine-thoracic aortogram. Enlargement of a 35 mm. frame. The injection is performed with the catheter in the left ventricle. The study demonstrates an enlarged left ventricle, which is also somewhat trabeculated. About 2 cm. above the valve, there is an area of marked narrowing of the aorta, which is hypoplastic above the site of stenosis. The coronary arteries are huge. Stenosis of the origin of the left common carotid, as well as minimal narrowing of the proximal portion of the innominate artery, is also visible. The descending thoracic aorta is normal in caliber. (D) Cine-pulmonary arteriogram. Enlargement of a 35 mm. frame. Profound stenosis is visible at the origin of a number of branch pulmonary arteries, particularly on the right, but on the left as well.

acteristics of the murmur and thrill and in which the diagnosis was suspected on clinical examination.

Merritt *et al.*<sup>48</sup> have studied 8 families with 1 or more affected individuals and found 25 probable cases in 4 of the 8. Pedigree analysis of familiar cases indicated an autosomal dominant mode of inheritance, with patients falling into two groups: (1) Sporadic cases with the complete syndrome

of supravalvular aortic stenosis, mental retardation, abnormal facies and peripheral pulmonary artery stenosis in which an extra chromosome may be present, producing a mosaic pattern; and (2) familial cases in which the trait is transmitted as an autosomal dominant with variable expression and often lacking mental retardation and facial similarity.

Eberle and Beuren<sup>22</sup> have recently re-



ported a lack of any chromosome abnormality in their studies of 3 patients with supravulvar aortic stenosis and low IQ's.

A familial influence has been suggested by Wooley *et al.*<sup>66</sup> who discovered supravulvar aortic stenosis in 2 siblings, and by Sissman *et al.*,<sup>56</sup> who described aortic stenosis in a patient whose grandmother was found to have supravulvar aortic stenosis at autopsy. The predominant occurrence of supravulvar aortic stenosis in males suggests an hereditary factor.

Recently, an association between supravulvar aortic stenosis and idiopathic hypercalcemia of infancy has been pointed out by Garcia *et al.*<sup>30</sup> A characteristic "elfin" facial appearance has been noted in patients with idiopathic hypercalcemia during infancy. Black and Bonham-Carter<sup>9</sup> have described some cases of supravulvar stenosis in older children with elfin-like facies who revealed histories of illness in infancy suggesting hypercalcemia. Serum calcium levels were normal in our own patients in whom it was obtained.

An abnormality of vitamin D metabolism has been postulated as the cause of hypercalcemia. If hypercalcemia is truly an integral facet of the supravulvar aortic stenosis syndrome, it must still be established whether the hypercalcemia is one component of a genetically determined multiple system abnormality, or whether the total syndrome stems from a derangement of vitamin D metabolism which may even be operative *in utero*. It has been shown by Eisenstein and Zeruolis<sup>26</sup> that, in animal studies, excess vitamin D has produced changes in the wall of the aorta, coronary arteries and heart valves ranging from subendothelial edema to calcification. Clinically, Rashkind *et al.*<sup>55</sup> have studied a large series of patients with idiopathic hypercalcemia of infancy and have noted a significant murmur in 63 per cent of 54 cases. They feel that the murmur is due to pathologic changes involving the cardiac valves and myocardium.

Why some patients with supravulvar aortic stenosis are not mentally retarded

and do not have the characteristic facies or peripheral pulmonary artery stenoses is, of course, not clear. Only 1 of our patients had altered facies and mentality. When Buchs and Wyler<sup>13</sup> reviewed the intelligence level of many previously reported patients, they found normal mentality in 27 of 45 cases. Of those classified as normal, 18 were boys and 8 girls. In an additional normal patient, the sex was not stated. The mentally deficient group was composed of 6 boys and 12 girls. Prior to the recognition of this syndrome, mental retardation was noted to be frequently associated with aortic coarctation. The absence of mental retardation in some cases of supravulvar aortic stenosis as well as the relative infrequency with other types of aortic stenosis indicates that it is not a simple consequence of aortic obstruction.

## 2. CLASSIFICATION

All of the previously reported cases of supravulvar aortic stenosis may be classified according to the system developed by Taybi and his co-workers:<sup>59</sup>

- Type I. Hypoplastic ascending aorta
  - IA. Tubular narrowing of the entire ascending aorta
  - IB. Narrowing more marked in the proximal region
- Type II. Normal or dilated ascending aorta with stenosis immediately above the aortic valve leaflets due to an internally protruding circumferential ridge and/or an externally detectable narrowing of the aorta above the sinuses of Valsalva.
- Type III. A thin membranous diaphragm

## 3. PATHOLOGY

Most cases fall in Taybi and co-workers Type I or II, with a definite localized area of narrowing just above the aortic valve and/or hypoplasia of the entire ascending aorta. Rarely, Type III, consisting of a true diaphragm, may be clinically significant.<sup>15</sup>

According to Kreel *et al.*,<sup>40</sup> the local stenotic area in Types I and II is produced by a ridge of connective tissue, of congenital origin, in which there is an overgrowth of elastic fibers. Study of the same area in normal hearts reveals that a small invagination of the aortic wall is normally present at the upper limit of the sinus of Valsalva. The supravalvular lesion may be a developmental exaggeration of this normal structure. Microscopically, the circumferential ridge has shown evidence of medial hypertrophy with focal disorganization and intimal fibrous proliferation.

The pathology of this lesion has been well described by Perou.<sup>54</sup> He has pointed out that "microscopically, aortic coarctation and supravalvular aortic stenosis are very similar. The basic microscopic lesion of true supravalvular aortic stenosis seems to be an angulation and exaggerated infolding with thickening and focal disorganization of the media capped by a zone of intimal thickening or hypertrophy showing various degenerative changes." The area of medial thickening is composed primarily of collagen and hyalinized fibrous tissue with a varying amount of elastic tissue as well. In some areas, there may be disruption of the elastic fibers. Variable degrees of intimal abnormality are also present and in some areas it is difficult to recognize the intimal tissue proper while in others there may be thickening due to fibrous proliferation. At the base of the infolding, several lacunae may be visible, lined by flattened endothelial-like cells. Foci of necrosis, calcification and, rarely, round-cell infiltration may also be seen in the protruding, stenosing ring. The stenotic site may serve as a focus for bacterial endocarditis, the residual fibrosis leading to progression of narrowing and eventually cardiac failure. This occurred in a case reported by Spencer and his co-workers.<sup>57</sup>

Abnormality of the aortic valves is frequently associated with the supravalvular lesion—in 25 per cent of cases, according to Edwards.<sup>24</sup> The valves may be bicuspid, and various degrees of thickening

and fibrosis of the leaflets can be present. The free edge of all or part of one or more cusps may form adhesions to the aorta at the site of stenosis, resulting in aortic insufficiency or partial to complete obstruction of the involved coronary artery.

The coronary arteries are usually markedly dilated and tortuous since they arise proximal to the stenosis and are perfused at a high pressure head. According to Williams *et al.*,<sup>64</sup> this high pressure may allow better toleration of the lesion than that noted when obstruction is at or below the valve. They point out, however, that the myocardial oxygen consumption and left ventricular hypertrophy increase in direct proportion to the systolic pressure load, making surgical relief of the obstruction an eventual requirement for survival. Morrow *et al.*<sup>49</sup> emphasize that subjecting the coronary arteries to such a high perfusion pressure makes the prognosis relatively grave, since the additional coronary flow apparently does not compensate for the increased burden on the left ventricle. In the experience of Neufeld *et al.*,<sup>52</sup> the intracoronary hypertension has led to marked thickening of the media associated with deposition of new elastic fibers in this area. Significant pathological changes have been found in the coronary vessels of patients in the first decade of life.<sup>24</sup> A stenosing process similar to that in the aorta may be found in the pulmonary artery branches and in the brachiocephalic vessels. The pathologic changes in the segmentally narrowed pulmonary vessels show abundant fibrous intimal proliferation. Distal to the stenoses, some vessels are dilated, with loss of elastic fibers in the media, while others remain narrow due to continuation of the intimal thickening.

#### 4. CLINICAL FINDINGS

*History.* Many patients are asymptomatic and are originally studied after detection of a murmur. Symptoms, if present, may range from mild or moderate exertional dyspnea to angina pectoris and congestive heart failure. All 4 of our patients



had symptoms, including fatigue, shortness of breath, fainting, dizzy spells, and syncope.

Eisenberg *et al.*<sup>25</sup> have noted that all patients whom he reviewed in 1964 were white and predominantly males. Although the age of patients varied from 7 to 68 years, the majority were in their teens. Mental retardation and physical underdevelopment are frequently found. Prematurity and low birth weight have been common but not consistent features, and failure to thrive has often been the only complaint.

*Physical Appearance.* Several authors have noted a distinctive facial appearance of patients with supraaortic stenosis and mental retardation.<sup>7,8,9,10,28,42,59,64,65</sup> This is often so characteristic that a bedside diagnosis can be made.

Resemblance of these patients to one another and dissimilarity to their own family members are striking. The facial resemblance seems derived largely from soft tissue similarity since no common abnormalities were found in roentgenograms of the skull reviewed by Williams *et al.*<sup>64</sup> The face is usually full with a broad forehead, widely set eyes, heavy cheeks, wide mouths, pouting lips, pointing of the chin, and prominent ears. The dental defects include generalized hypoplasia of the teeth, particularly the upper, middle incisors, malformations of the deciduous molars, partial anodontia, and late mineralization.<sup>8</sup> The mandible is hypoplastic, with a disparity in ramus size of the small horizontal and the large vertical rami. This is partially responsible for the peculiar facial appearance.

The temperament of these patients is usually friendly and they are active, happy and talk with a deep metallic voice.

*Physical Examination.* In distinguishing supraaortic stenosis from other types of congenital aortic stenosis, a few diagnostic clues are helpful:

1. A systolic ejection murmur and systolic thrill are frequently louder in the neck and suprasternal notch than in the right first or second intercostal space, although not invariably.

2. None of our patients had aortic systolic ejection clicks, nor are these reported by others. The aortic second sound is usually normal. Occasionally, the associated murmur of aortic insufficiency may be heard, indicating that one or more of the leaflets are involved in the deformity.

3. Discrepancy in the pulses and blood pressures in the arms may be noted due to associated obstructive lesions in the great vessels.

*Electrocardiogram.* The electrocardiogram does not offer any identifying characteristics. As in other types of valvular and subvalvular disease, there may be very high pressure gradients and only a small degree of left ventricular preponderance, indicating a poor relationship between degree of left ventricular hypertrophy and the pressure gradient. In some cases, associated mitral insufficiency and cardiomyopathy may also influence the electrocardiogram. Generally, a left ventricular or combined hypertrophy pattern is identified, depending upon the presence or absence of pulmonary stenoses. Three of our 4 patients had a left ventricular hypertrophy pattern, and 1 had a normal electrocardiogram.

#### 5. ROENTGENOLOGIC FINDINGS

Gross cardiac enlargement was not present in any of our cases, although there was a slight increase in size in 1. The left ventricle was somewhat prominent in 2 cases, and the right ventricle was noted to be slightly enlarged in the case with multiple pulmonary branch stenosis. A striking finding was the absence of poststenotic dilatation in all cases, and the small size of the aortic knob. The lung fields were normal. Fluoroscopically, aortic pulsation was feeble.

The absence of poststenotic dilatation serves as a useful, although by no means infallible, differentiating point from valvular aortic stenosis. An analysis of 121 cases, in 70 of which roentgenograms were reproduced or descriptions included, indicated that absence of a prominent ascending aorta and a small aortic knob were noted in over 90 per cent of cases. Sixty per cent

had slight to moderate cardiac enlargement, and the remainder was normal. Discrete left ventricular enlargement or prominence was thought to be present in 57 per cent of cases. In 10 per cent, left atrial enlargement was also noted. Coincident right ventricular enlargement was apparent in 14 per cent of cases, usually those with associated multiple branch stenoses. The irregular "sausage-like" appearance of the central branches of the pulmonary arteries described in the presence of multiple branch stenoses was rarely recorded. This nodular, irregular vascular pattern is visible in the para-hilar areas in some cases, and is produced by localized narrowing and poststenotic dilatation.

#### 6. CARDIAC CATHETERIZATION

The physiologic findings in valvular, subvalvular, and supravalvular stenosis have been well described by Hancock.<sup>35</sup> It is important that the catheter side holes be as far distal in the catheter as possible when the stenotic segment is short. If the constriction is well beyond the valve, three distinct pressure zones exist: (1) High ventricular systolic pressure; (2) similarly high proximal aortic systolic pressure; and (3) distal to the stenosis, an abrupt fall in systolic pressure with relatively little change in diastolic pressure.

Catheter studies of the right heart may demonstrate pressure gradients across the stenotic pulmonary artery segments and elevated main pulmonary and right ventricular pressures.

#### 7. ANGIOGRAPHY

Contrast study of the thoracic aorta is by far the best method of determining the presence, location, and degree of supravalvular aortic stenosis. Our own approach is to utilize the percutaneous transfemoral Seldinger method for catheter insertion, injecting a bolus of contrast agent in the root of the aorta with cine or large film recording. Motion picture techniques are admirable for demonstrating the flow through the distended coronary bed, and, in addition, they afford better delineation of aortic valve

motion. Associated lesions such as fusion of valve leaflets can readily be identified. At the time of thoracic aortography, pressures are obtained at the ventricular, supravalvular, and suprastenotic area.

Preoperative study is essential in order to determine the presence or absence of hypoplasia of the aorta above the site of stenosis to allow the surgeon to plan his approach, and also to obtain some estimate of the operative risk.

In all of our cases, the supravalvular narrowing was localized to the site just above the sinuses of Valsalva. In 2 instances, diffuse hypoplasia of the ascending aorta accompanied the stenosis. The sinuses of Valsalva were large, and gross dilatation of the coronary arteries was a prominent feature. The valve leaflets were normal. Similarly, no abnormalities of the pulmonary arteries were visible except in 1 instance, in which multiple pulmonary artery stenoses were found.

Angiocardiographic studies were performed in 69 cases recorded in the literature. In two-thirds of these, the injection was made into the left ventricle, and in the remaining one-third a supravalvular injection was performed. A few patients were studied following injection into the right cardiac chambers. Cine-recording was employed in one-quarter of cases, and large film recording in three-fourths.

Left ventricular enlargement was noted in about 25 per cent of cases, whereas hypertrophy was rarely described, although obviously present in some cases. Abnormal limitation of movement of the aortic valve leaflets was apparent in 10 per cent of cases. The appearance of the aorta was very similar in most cases, with a localized segment of narrowing immediately above the sinuses of Valsalva, or within 1-2 cm. beyond. Above the localized segment, the aorta was of normal size in 60 per cent of cases, hypoplastic in 30 per cent, and dilated in 10 per cent. In 3 cases, there was hypoplasia of the entire aorta. The stenosis was mild in 21 per cent, moderate in 56 per cent and severe in 17 per cent. The length of the narrowed segment varied between 0.5 cm. and 3.0 cm.



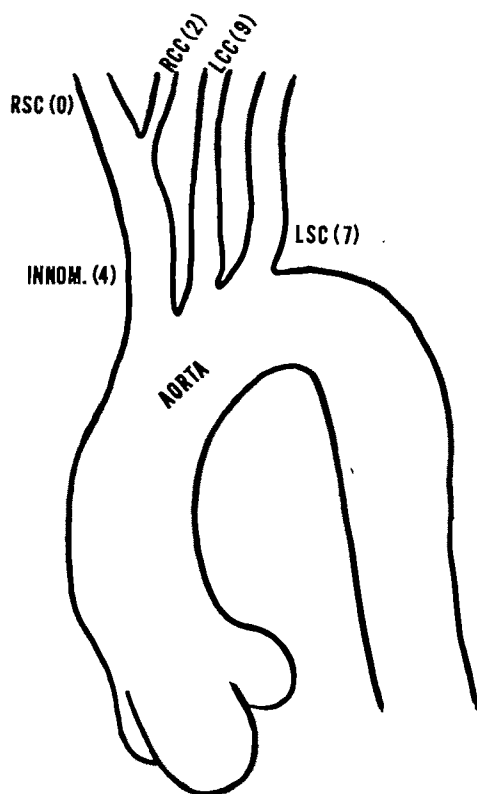


FIG. 5. Brachiocephalic branch stenosis in supravalvular aortic stenosis. Incidence and location in 22 cases out of a total of 121 reported in the literature. Figures in parentheses indicate incidence of stenosis of each vessel.

Among 35 cases in which pulmonic stenosis coexisted, it involved branches on both sides in 57 per cent, one side in 20 per cent, the main pulmonary artery in 9 per cent and the pulmonic valve in 14 per cent.

#### 8. BRACHIOCEPHALIC BRANCH STENOSIS

Occasional cases have been reported in which stenotic lesions have been observed in the brachiocephalic vessels.<sup>10,24,29,46,66</sup> Often, suspicion of such a finding was first evident when a pulse or blood pressure differential was noted during the physical examination. This was true of 2 of our own cases in which stenosis was evident. The fact that 3 of our 4 cases demonstrated some degree of narrowing of one or more of the branch vessels of the aortic arch strongly indicates that the incidence of this associated anomaly is higher than has been

reported. A review of 121 published cases showed a relatively small number of localized brachiocephalic lesions, but careful analysis of the roentgenographic illustrations indicated that some lesions were not alluded to.<sup>28,38,53</sup> Furthermore, in many of the cases previously recorded in the literature, bilateral upper extremity blood pressures were not reported, and may well have failed to reflect stenosis which was present.

An analysis of the location of brachiocephalic stenosis in supravalvular aortic stenosis indicates the distribution shown in Figure 5.

Wooley *et al.*<sup>66</sup> have reported a series of 4 patients, all of whom had pulse and blood pressure differences in the upper extremities. Angiographic studies revealed a discrepancy in the size of the subclavian artery diameter on the side with the decreased pulse or blood pressure, in spite of the fact that local stenosis was not visible. Lurie and Mandelbaum<sup>44</sup> suggested that anatomic stenosis may not always be necessary, since turbulence beyond the area of supravalvular stenosis may result in interconversion of vascular potential and kinetic energy, leading to the asymmetrical physical findings. Others have supported this point of view.

The pathologic changes in the branch stenotic lesions have not been studied in detail. Edwards has reported 1 case in an 11 year old patient showing gross thickening of the media with associated narrowing of both the left common carotid and innominate arteries. These findings must be distinguished from such lesions as Takayasu's arteritis, which is characterized by chronic progressive inflammatory change leading to fibrosis and thrombosis, with eventual obliteration of the vessel lumen.<sup>34</sup> Atheromatous plaques and secondary calcification may obscure the pathological differences in the final stages. In Takayasu's disease, changes have been reported in other vessels such as the abdominal aorta and peripheral arteries. Stenotic lesions of vessels other than the aorta, the brachiocephalic, and the pulmonary vessels should also be carefully

sought in patients with supravalvular aortic stenosis.

#### 9. SURGICAL MANAGEMENT

The surgical approach to supravalvular aortic stenosis has recently been reviewed by several authors.<sup>16,18,40,46,53,58</sup> The recommended technique includes a linear aortotomy through the stenotic area and into the noncoronary sinus. Following this, a diamond or teardrop-shaped teflon prosthesis is sutured in place. At surgery, the stenosis may be much more severe than is observed on the exterior aspect of the aorta due to a massive fibrotic ring impinging upon the aortic lumen. The presence of valvular deformities makes surgery more difficult and hazardous. Excellent 3 year postoperative results have been demonstrated angiographically by Nordström and Silander.<sup>53</sup> In the presence of a significant gradient, surgical therapy is indicated and may be expected to be highly successful.

Although complications may arise in these cases with peripheral pulmonary artery stenosis, such as hemorrhage from the dilated poststenotic segments following arteritis or necrotizing lung infection, surgery has generally not been feasible, to date.

#### SUMMARY

Supravalvular aortic stenosis is frequently associated with other congenital abnormalities such as pulmonary artery branch stenosis, unusual facies, retarded physical development, abnormal dentition, and mental retardation. Stenosis of the origins of the brachiocephalic vessels may be found in a significant percentage of cases. Its presence should be suspected when asymmetrical pulses or blood pressures in the upper extremities are found on physical examination, and may be verified by thoracic aortography.

Among the 4 new cases of supravalvular aortic stenosis reported herein, 3 had brachiocephalic vessel stenosis. All were diagnosed by roentgen contrast study and pressure measurements, with surgical con-

firmed and successful surgical therapy in 3.

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## CATHETER POSITION: AN AID IN DIAGNOSIS OF DISSECTING ANEURYSM OF THE THORACIC AORTA\*

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IN MOST cases, the diagnosis of a dissecting thoracic aneurysm can be clearly established by aortography. In some instances, however, particularly in dissecting aneurysms of some duration, it may be difficult to distinguish the true from the false channel, angiographically thus having the appearance of a normal aorta. Under these circumstances, the position of the catheter during the injection of contrast medium proved to be most important in establishing the correct diagnosis.

In 1 of our patients who underwent aortic valve replacement for aortic insufficiency, an old dissecting aneurysm was found at the time of surgery which had not been recognized preoperatively. A review of the retrograde aortogram showed a markedly dilated ascending aorta with a wide annulus and aortic insufficiency quite characteristic of Marfan's syndrome. The entire aortic arch opacified uniformly and simultaneously with a false channel (Fig. 1), which was not appreciated. On review of the aortogram, it was noted that the catheter did not hug the outer wall during the injection, which should have suggested the correct diagnosis. An identical observation made in an additional patient warranted this report (Fig. 2).

### DISCUSSION

A dissecting aortic aneurysm is one of the primary differential diagnostic considerations in patients presenting with severe chest pain. The dissection occurs most commonly at the following sites: low in the ascending aorta just above the posterior aortic valve cusps near the left coronary

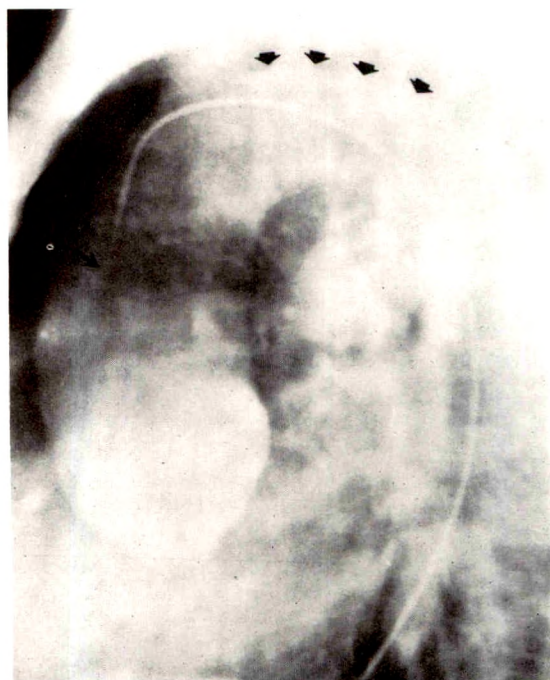


FIG. 1. Lateral aortogram showing diffuse dilatation of the ascending aorta consistent with cystic medial necrosis. Note film exposed during injection as indicated by contrast medium being ejected from the catheter tip (single arrow). Separation of catheter and distal aortic arch indicating dissection (multiple arrows).

artery and just distal to the origin of the left subclavian artery.<sup>1,3,4</sup> The dissecting intramural hematoma invariably progresses in a centrifugal manner in the region of the aortic arch. The hematoma, therefore, lies superiorly in the mid arch and posteriorly and to the left in the distal aortic arch (Fig. 3). In the region of the descending thoracic aorta and abdominal aorta, the dissection is also most commonly found posteriorly and to the left, with a

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higher incidence of dissection into the left renal artery and left iliofemoral system. The reason for progression of the dissecting hematoma along the superior posterior contour of the aortic arch is not definitely known but very likely related to the simple physical principle of preservation of momentum (Fig. 4). The same physical principle is also responsible for straightening of the catheter along the greater curvature of the aorta during the injection of contrast medium since the flow tends to progress in a straight direction.

In order to evaluate this phenomenon, catheters of various designs were placed into a curved polyvinyl tube simulating the aortic arch. During the injection of contrast medium with a power injector, the catheter invariably rose to the greater curvature of the simulated aorta (Fig. 5A) and resumed its original position after completion of the injection (Fig. 5B). The momentum of the bolus, injected with pressures commonly used in aortography, tends to straighten the curvatures of the catheter, as is known from well established laws of hydrodynamics.

In order to check the validity of this

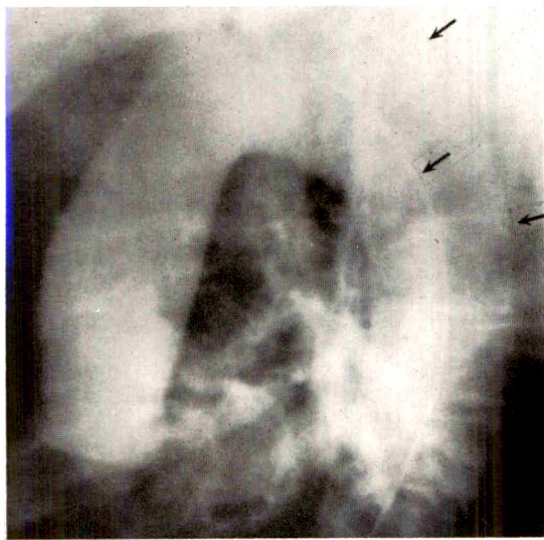


FIG. 2. Lateral aortogram showing wide separation of catheter and aortic contour in the posterior arch (arrows), being diagnostic of large dissection provided film is exposed during the injection phase.

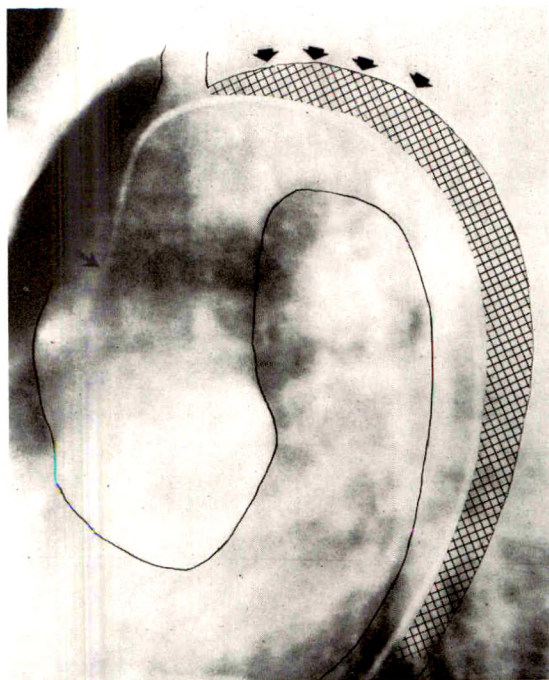


FIG. 3. Same patient as in Figure 1. Explanatory diagram showing the invariably posterior location of the dissecting hematoma in the posterior aortic arch.

phenomenon in clinical cases, 100 aortograms of patients without dissecting aneurysms were reviewed and the measured dis-

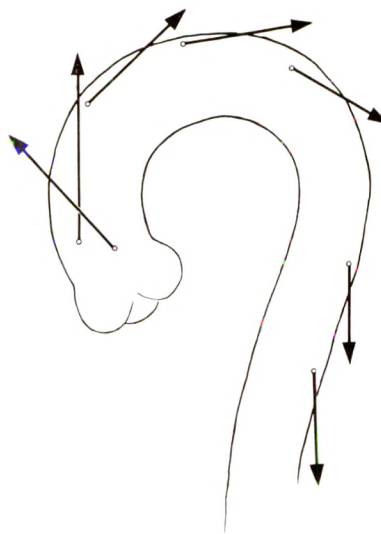


FIG. 4. Diagram showing areas of thoracic aorta to which forces of momentum are directed and coinciding with the common routes of dissection.



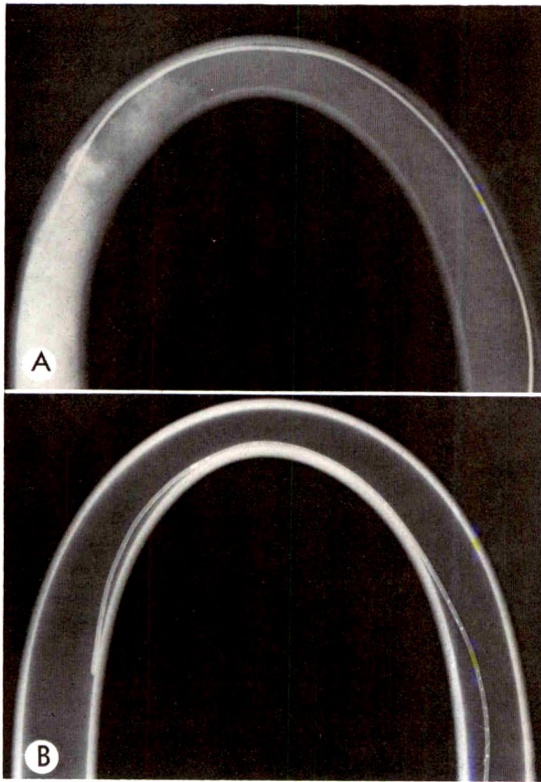


FIG. 5. (A) Lateral roentgenogram of aortic phantom during injection of contrast medium. Catheter seen to closely hug outer wall. (B) After completion of injection, catheter falls away from outer surface.

tance between the catheter and the inner margin of the posterior aortic arch on the lateral film studies during the injection was 2 mm. and did not exceed 4 mm. In rare instances of ectasia or coarctation of the aorta (Fig. 6), the catheter position cannot be relied upon. Furthermore, only aortograms in lateral projection during the power injection of contrast medium can be used, since the catheter tends to fall away from the aortic wall following completion of the injection.

The angiographic appearance of dissecting aneurysm may vary considerably depending upon the degree of blood flow in the dissecting hematoma. In dissecting aneurysms which have not re-entered or where re-entry into the true lumen is small, blood flow in the false channel may be absent or very slow. This results in a typical

angiographic appearance with nonopacification of the false channel on early film studies and more or less good visualization of the false channel on delayed films. The differential contrast density in the true channel as compared to the false channel is usually quite striking and has been well discussed in a recent paper on this subject by Eyler and Clark.<sup>2</sup> In patients with "healed" dissections wherein re-rupture and complete endothelialization of the false channel has occurred resulting in a "double barrel" or "triple barrel" aorta, blood flow in the true and false channel may be of almost equal rate. Consequently, there may be immediate opacification of both channels

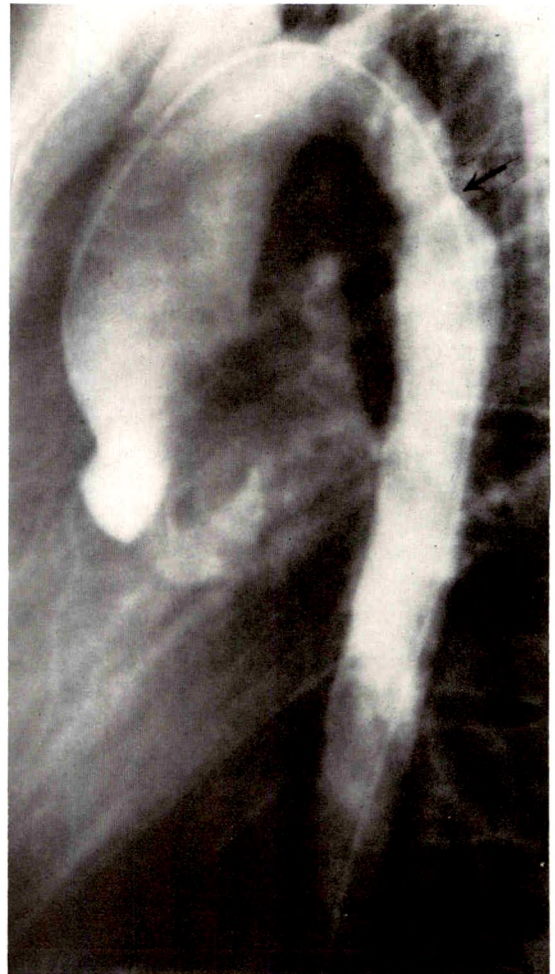


FIG. 6. False separation of catheter and aortic margin due to the presence of coarctation (arrow). Note also bicuspid aortic valve.

which are separated only by the dissected media. If the dissected media is very thin as in cystic medial necrosis, the angiographic diagnosis of dissecting aneurysm may be extremely difficult because the very thin dissected media is usually visualized only as a fine radiolucent membrane, thus being easily overlooked. The unusual position of the catheter during the injection of contrast medium as seen on the lateral aortogram, however, is invariably present and the diagnosis can be firmly established.

#### SUMMARY AND CONCLUSION

In cases of recent dissecting aneurysms, blood flow in the false channel is markedly slowed, thus resulting in nonopacification or delayed contrast opacification of the dissecting hematoma. Dissecting aneurysms which have re-ruptured into the true lumen or so-called "healed" dissecting aneurysms may have a "double barreled" aorta with two or sometimes three endothelial lined channels. The speed of blood flow in various channels may be almost equal, resulting in simultaneous opacification of the true and false channels and giving the erroneous angiographic appearance of a normal aorta. The dissected media may be very thin, particularly in patients with cystic medial

necrosis of the aorta. Under those circumstances, the unusual position of the catheter during the injection of contrast medium may be the only diagnostic clue, as observed in 2 of our patients. The reliability of this sign was studied in a model and evaluated in 100 lateral aortograms. The distance between the catheter and the inner border of the superior and distal aortic arch as seen on the lateral film study was measured as 2 mm. and did not exceed 4 mm. The sign is only valid on lateral aortograms during the period of injection.

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# CALCIFIED AND DILATED ASCENDING AORTA DUE TO ATHEROMATOUS OCCLUSIVE DISEASE SIMULATING COARCTATION OF THE AORTA\*

## A REPORT OF A CASE AND THEORY OF PATHOGENESIS

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**B**ECAUSE of its unusual nature, a patient with a calcified and dilated ascending aorta due to atheromatous occlusive disease and simulating coarctation of the aorta is reported and a theory of the pathogenesis of the case is presented. A similar case was reported recently by Munasinghe and Rajasuriya.<sup>9</sup>

### REPORT OF A CASE

A 21 year old Caucasian woman (N.Y.H. No. 608983) referred by Dr. Horace S. Baldwin, was admitted on September 10, 1951, with a complaint of high blood pressure. Two years prior to admission, while being examined for painless enlargement of lymph nodes (eventually diagnosed as infectious mononucleosis), the blood pressure had been 170/80, and 2 months prior to admission it was 190/80 mm. Hg. The patient had had recurrent bilateral headaches associated with sinusitis up to the age of 14 years; during the past 7 years these had subsided. Rhinitis, worse in the fall, was her only other complaint.

Physical examination showed a well developed and well nourished woman in no discomfort. Her weight was 132 pounds and her height, 5 feet, 8 inches. The lungs were clear. The heart was not enlarged. A systolic murmur of moderate intensity was heard over the entire precordium, loudest at the second intercostal space along the left sternal border. The femoral pulses were diminished. Intercostal and inter-scapular arterial pulsations were not evident. The blood pressure in the extremities was: right arm, 165/65; left arm, 170/65; right leg, 108/75; and left leg, 140/80 mm. Hg. Both feet

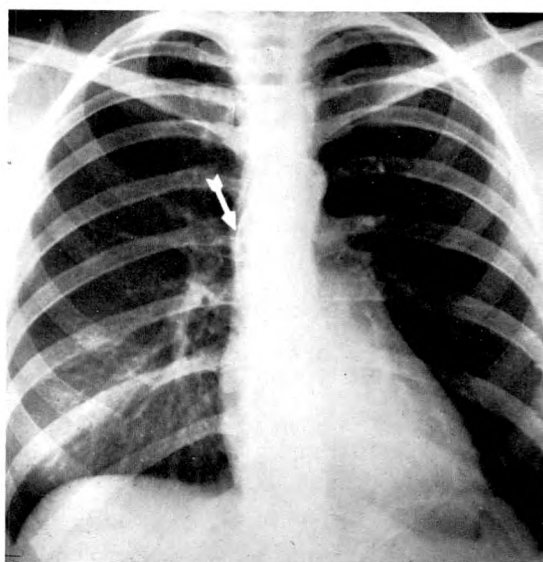


FIG. 1. Teleroentgenogram of the chest showing marked calcification of the ascending aorta and arch (arrow). The left ventricle is also slightly enlarged.

were cool and had diminished pulses. Lymphadenopathy was not found. The electrocardiogram showed normal sinus rhythm, rate of 88 per minute, and no deviation of the electrical axis. Urinary and blood studies were normal. Serologic tests for syphilis were negative.

Roentgenograms of the chest demonstrated rounding of the left border of the heart, marked calcification and dilatation of the ascending aorta, a small aortic arch, and narrowing and calcification of the descending aorta just below the arch (Fig. 1 and 2). Angiocardiograms made on September 13, 1951, showed normal cardiovascular structures except for slight enlarge-

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ment of the left ventricle, dilatation of a calcified ascending aorta, and narrowing of the descending aorta below the left subclavian artery (Fig. 3). The ascending aorta measured 40 mm. in diameter (upper limit of normal is 38 mm.), the area beyond the origin of the left subclavian artery measured 7 mm., and the descending aorta was 25 mm. The ascending aortic calcification ended at the arch and did not involve the descending thoracic aorta. There were no collateral arteries.

Simultaneous and direct arterial blood pressure determinations showed: left brachial artery, 180; left femoral artery, 110 mm. Hg. Abdominal roentgenograms did not reveal calcification. Abdominal aortograms and pyelograms showed normal kidneys and collecting system. Operation was not advised and the patient was discharged on September 24, 1951.

A communication from Dr. Albert A. Kattus, Jr., on March 27, 1958,<sup>8</sup> related that the patient had been operated on at the University of

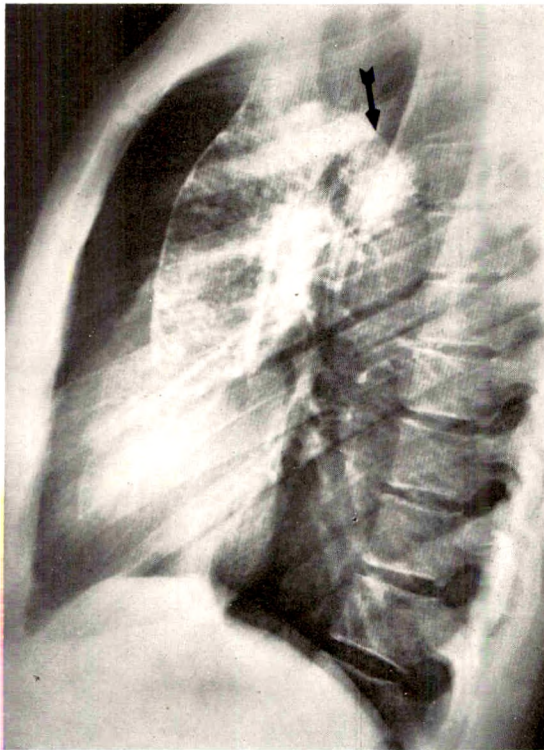


FIG. 2. Lateral roentgenogram of the chest shows to advantage the dilatation and marked calcification of the entire ascending aorta. The aortic arch and the top of the descending aorta are also calcified. Note the narrowed lumen of the descending aorta (arrow).

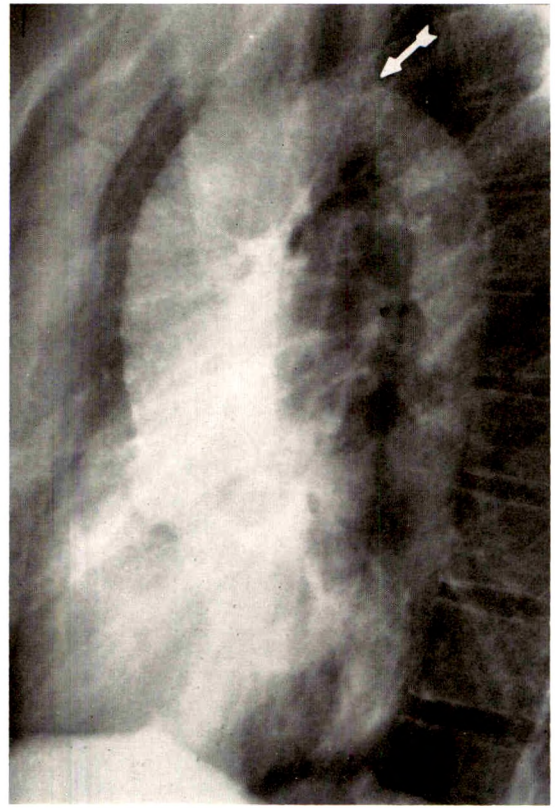


FIG. 3. Left anterior oblique angiogram made in 1951 shows the dilated ascending aorta and narrowed descending aorta just beyond the origin of the left subclavian artery (arrow). Note that the internal mammary arteries are of normal size and that other collateral arteries are absent.

California Medical Center, Los Angeles, California. Subsequently, in 1959, her case was published.<sup>5</sup> She had been admitted to the University of California Hospital in October, 1957, complaining of high blood pressure which measured 240/70 in the arms and 110/75 mm. Hg in the legs. The femoral pulses were barely palpable. A blowing systolic (Grade 4) murmur was heard over the precordium and was transmitted into the neck and axilla. An early diastolic (Grade 2) murmur was audible along the left sternal border in the fourth interspace. The electrocardiogram showed left ventricular hypertrophy. The roentgenogram of the chest showed moderate enlargement of the heart and dilatation and severe calcific atherosclerosis of the ascending aorta with narrowing of the descending aorta beyond the subclavian artery.

On January 10, 1958, thoracotomy and exploration of the thoracic aorta revealed a dilated



calcified ascending aorta which began at the aortic valves and reached 1 cm. distal to the subclavian artery. There was narrowing of the descending aorta to one-third of its proximal diameter at that point with only a faint pulsation distally. Aortotomy 1 cm. beyond the origin of the left subclavian artery revealed stenosis with almost total obstruction of the aortic lumen by atheromatous calcific deposits. The obstructing calcified material was resected for a short distance, leaving a lumen of 1.5 to 2.0 cm. in diameter. Because the endarterectomy segment was very thin, the aortotomy was resutured and reinforced with an aortic homograft.<sup>5</sup> After surgery, simultaneous blood pressures of the arms and legs were 250/78 and 245/80 mm. Hg, respectively. One year post-operatively, the blood pressure of the arm was 158/54 and in the leg it was 180/100 mm. Hg.

Recent communication on September 28, 1965, with Dr. Albert A. Kattus, Jr.,<sup>8</sup> disclosed that the patient had had yearly examinations since 1958, the last one very recently. Two years ago she had had mild congestive heart failure which was alleviated by diuretic therapy. In the same year, she had symptoms of basilar artery insufficiency with slight visual aberrations and paresthesias of the inside of the mouth. Angiography demonstrated the left subclavian steal syndrome.<sup>12</sup> This was attributed to constriction of the left subclavian artery at its origin, secondary to clamping at the time of surgery.<sup>8</sup> She has been asymptomatic during the past year. Treatment has included digitalis, low salt diet, and an occasional dose of a diuretic drug. She has been able to do her housework which included the care of her children. Physical examination showed gross insufficiency of the aortic valve; the blood pressure in the arms was 190/80 mm. Hg.

#### DISCUSSION

Because of the unusual dilatation and calcification of the ascending aorta and absence of collateral arterial vessels, the diagnosis of ordinary (congenital) coarctation of the thoracic aorta was questioned following angiographic studies at this center in 1951. Despite this, 4 years later, she was included among a series of patients who were listed with contraindications to surgical therapy of coarctation of the aorta.<sup>10</sup> Operation was believed to be inad-

visable because of the severe atheromatous disease of the aorta. Seven years later, after persistence of hypertension and the development of aortic insufficiency, aortotomy at another hospital disclosed calcified atherosclerotic thrombotic disease severely occluding the aorta below the origin of the left subclavian artery.<sup>5</sup> Six years later, the aortic insufficiency and hypertension persisted. A bout of heart failure, cerebral insufficiency, and subclavian steal syndrome suggest progression of the disease.

Atheromatous thrombotic occlusive disease of the descending aorta is not as common as abdominal thrombotic occlusive disease.<sup>14,17</sup> Usually, the latter occurs in middle-aged patients. In recent years, however, many reports of premature atheromatous disease of the thoracic aorta, occurring especially in young women, have appeared in the literature.<sup>2,3,7,11</sup> Takayasu<sup>16</sup> in 1908 called attention to occlusive aortic arch disease (pulseless disease) in Japanese women and, since then, other cases in Orientals<sup>3</sup> and also in Caucasians in both men and women have been reported.<sup>2</sup> In these cases the calcifications were usually limited to the aortic arch and histologically had the appearance of arteritis.

Gross<sup>6</sup> in 1945 and Smith *et al.*<sup>12</sup> in 1965, in reporting gangrene of the extremities in the newborn and infants, raised the possibility of emboli derived from closure of the ductus arteriosus playing a role in the disease. Can it be that such a mechanism initiates atheromatous occlusive disease beginning in the infundibulum of the aorta? It is conceivable that instead of the calcific plaque in the aorta that Dalith<sup>4</sup> has described at the site of the closure of the ductus, the thrombus continues to grow, attracts more clot causing atheromatous and arteritis changes,<sup>1</sup> and, finally, occludes the lumen of the aorta producing the signs of coarctation of the aorta and hypertension. Just as patency of the ductus arteriosus predominates in the female sex, so does atheromatous occlusive disease of the descending aorta occur predominantly in young women.<sup>2</sup>

## SUMMARY AND CONCLUSIONS

The case of a 21 year old woman with hypertension, calcification, dilatation of the ascending aorta, and partial occlusion of the descending aorta just beyond the left subclavian artery simulating congenital coarctation of the aorta is described. Aortotomy, 7 years later at the age of 28 years, revealed occlusion of the aorta due to calcified atheromatous thrombus. Within a 6 year period, extension of the disease into the aortic arch vessels probably caused basilar arterial insufficiency and the subclavian steal syndrome. To explain this phenomenon, it is theorized that instead of thrombosis, healing, and closure of the ductus soon after birth, the thrombus is propagated into the lumen of the aorta, attracts more clot, atheromatous material, and eventually causes premature thrombotic occlusive disease of the aortic arch and descending aorta.

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## ATHEROSCLEROSIS IN THE RABBIT VISUALIZED BY CARDIOVENTRICULOGRAPHY AND SUBTRACTION\*

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CINCINNATI, OHIO

**D**URING participation in a national co-operative study of cerebral vascular disease, arteriographic investigations showed the diffuse nature of the atherosclerotic process. Vascular surgery proved useful in some patients, but it became apparent that ultimate success in preventing strokes and other complications from atherosclerosis would stem from basic research into the prevention or regression of the atherosclerotic process. Rabbits have been used to study the effects of diets, drugs and procedures on atherosclerosis,<sup>2</sup> but until now it has been necessary to sacrifice the animals in order to carry out the investigations.

A safe, simple, repeatable, left cardioventriculography technique for demonstrating the patterns of atherosclerosis in rabbits has been devised. Previous investigators have used this technique in man and in animals with few complications.<sup>3,4,6</sup> Subtraction techniques, originally developed by Ziedses des Plantes,<sup>7</sup> were applied to our experimental angiography to enhance the details (Fig. 1, *A* and *B*; and 2, *A* and *B*).

With these techniques, excellent visualization was obtained of plaques in the aorta and its branches, and the carotid and subclavian arteries (Fig. 3; 4; and 5). In addition, poststenotic dilatations were observed (Fig. 3 and 4), as well as uncoilings of the aorta (Fig. 6). Arteriograms in normal rabbits failed to show these changes.

In the present work "plaques" will be referred to as "atherosclerotic" without any implication that the condition in rabbits is the same as in man.<sup>5</sup> However, the uncoilings of the aorta, the dilatation of

vessels, and the stenoses seen in the rabbit appear to be like those in man.

### ANIMAL PREPARATION

Rabbits were chosen for this investigation because they commonly are used in the study of atherosclerosis and because they are available in large numbers, are easy to handle, and are inexpensive. Twenty New Zealand white rabbits were used, evenly divided as to sex, and averaging 2,000 gm. each at the beginning of the study.

In developing the left cardioventriculography technique, 10 control rabbits were subjected to 75 studies over a 1 year period. After the technique was mastered, it was applied to 10 cholesterol-fed rabbits during the next year.

A 2 per cent cholesterol diet was prepared with crystalline cholesterol\* dissolved in ethyl ether. The ether solution was mixed with Albers Rabbit Family Ration† and allowed to evaporate, leaving cholesterol-impregnated pellets. The control rabbits received Albers Rabbit Family Ration *ad lib.* throughout the investigation.

### ANESTHESIA

The rabbits were given pentobarbital sodium (nembutal), 40 mg. per kg. body weight by intraperitoneal injection. Open ether was then supplemented, if additional anesthesia was needed prior to arteriographic injection, to prevent response to painful stimuli. The success of the subtrac-

\* Nutritional Biochemical Corporation, Cleveland, Ohio.

† Albers Milling Company (Division of Carnation Company), Los Angeles, California.

\* From Department of Neurology, University of Cincinnati College of Medicine, and Cincinnati General Hospital, Cincinnati, Ohio.  
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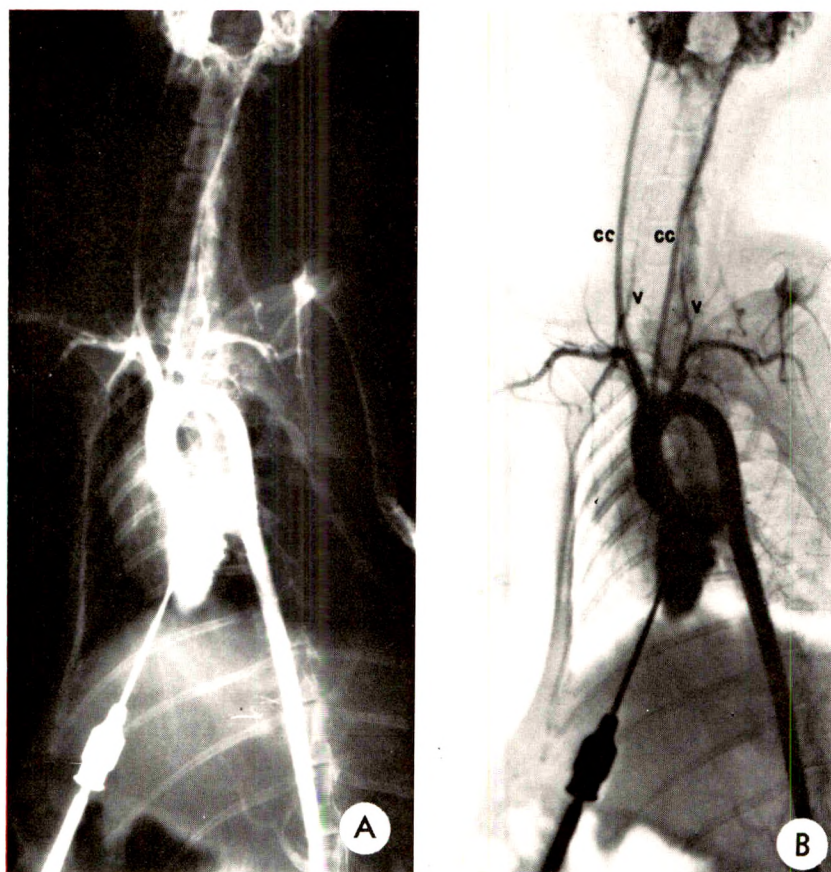


FIG. 1. (A) Normal rabbit. Anteroposterior view. (B) Subtraction of A demonstrating removal of bone shadows to outline vessels. CC=common carotid; V=vertebral.

tion technique depends on the subject being as motionless as possible during the procedure.

#### ROENTGEN TECHNIQUE

Equipment consisted of an Elema-Schönander film changer with a  $14 \times 14$  inch film and a 12:1 grid; rate of exposure was 4 frames per second for 3 seconds. Exposures were set between 70 to 80 kv. peak of 100 ma. for 0.1 second. Film focus distance was 36 inches; focus size was  $1.2 \times 1.2$  mm. Scout roentgenograms were always taken to check roentgenographic settings. Coning was used to reduce scatter radiation and to increase sharpness of detail. If available, a smaller focal spot with 3-phase equipment would increase detail and allow for magnification.

Serial study allowed constant visualization of the arterial system. However, a single injection could be done and would provide excellent detail of vessels if the timing of the injection was right.

It was necessary to take roentgenograms at different angles in order to view plaques. A plaque on the posterior wall frequently would not show in an anteroposterior view, but narrowing of the contrast medium column by plaque could be seen in a lateral or oblique view.

#### TECHNIQUE OF LEFT VENTRICULAR PUNCTURE

The rabbit was taped in a supine position on the roentgenographic table. The chest was centered. The xiphoid process was located by palpation and a small area was



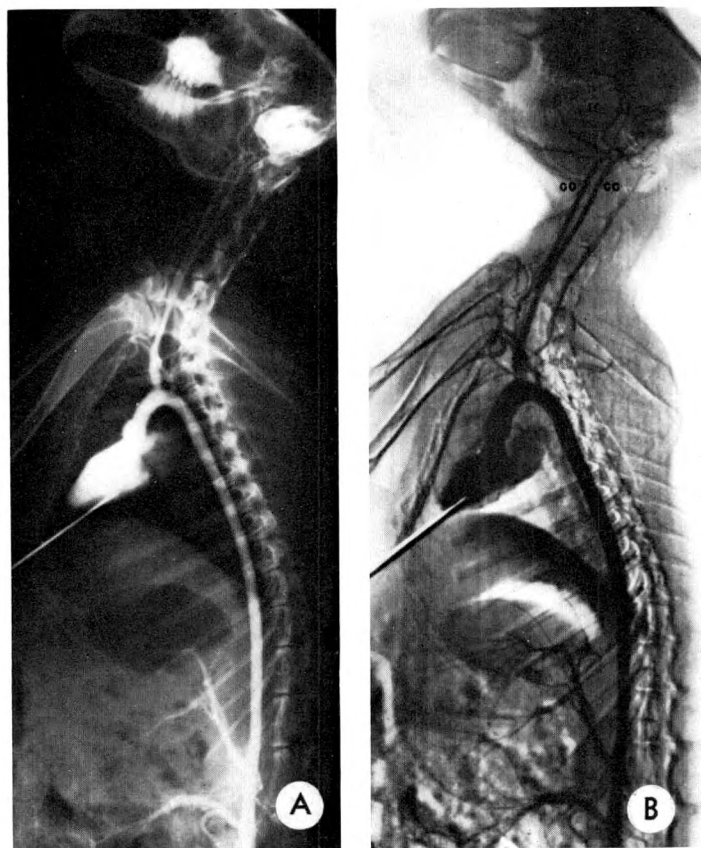


FIG. 2. (A) Normal rabbit. Lateral view of same rabbit as in Figure 1, A and B. (B) Subtraction of A showing increased detail of intracranial and vertebral vessels. CC = common carotid; IC = internal carotid.

cleaned with alcohol. A  $2\frac{1}{2}$  inch, No. 19 needle was connected to the male adapter of a 20 inch venotube (Abbott Company). The needle tip must be sharp and the edges of the tip rounded. The sharp point allows immediate penetration of the heart without major displacement. The rounded edges prevent laceration of the myocardium.<sup>3</sup> A 10 cc. syringe was connected to a Luer-Lok, 3-way stopcock, which then was attached to the female adapter of the venotube. The venotubing was filled with isotonic saline, freed of all air and closed by turning the stopcock. All connections were tightened prior to injection to prevent leakage of air into the system and to avoid possible separation of the injection assembly.

The needle and tube system was held with the right hand and the xiphoid process was palpated with the index finger of the left hand. The needle was inserted under the skin between the junction of the last right costal cartilage and the xiphoid pro-

cess. The venotube was then opened to permit immediate observation of entry into the ventricle. The needle was directed under the sternum toward the tip of the left shoulder and angled down 35 degrees. The needle was pushed toward the diaphragmatic surface of the heart until ventricular pulsations were felt against the needle tip. The needle then was advanced rapidly 5 mm. further. The appearance of bright oxygenated arterial blood in the venotube and a pulsating needle was evidence that the needle tip was in the left ventricular chamber. If dark unoxygenated blood was seen and the pulsation was weak, the right ventricle had been entered by mistake and the needle was withdrawn and redirected toward the left.

In the early studies, fluoroscopy with an image intensifier and mirror (Picker Company) was used to determine the location of the needle. Once the left ventricular puncture technique was mastered, fluoroscopy

was omitted. Needle placement also could be determined by a small injection of contrast medium and exposure of a single film with the needle still in place.

If the blood flow was slow or absent in the tubing, the needle was elevated or depressed to attempt to establish flow. If the puncture was still unsuccessful, the needle was withdrawn slowly and a second puncture was attempted. Only 2 attempts were made for each rabbit on any day to prevent excessive trauma. When flow was established, 1 to 2 cc. of isotonic saline solution was injected intermittently to prevent clotting, eliminating the need for heparin.

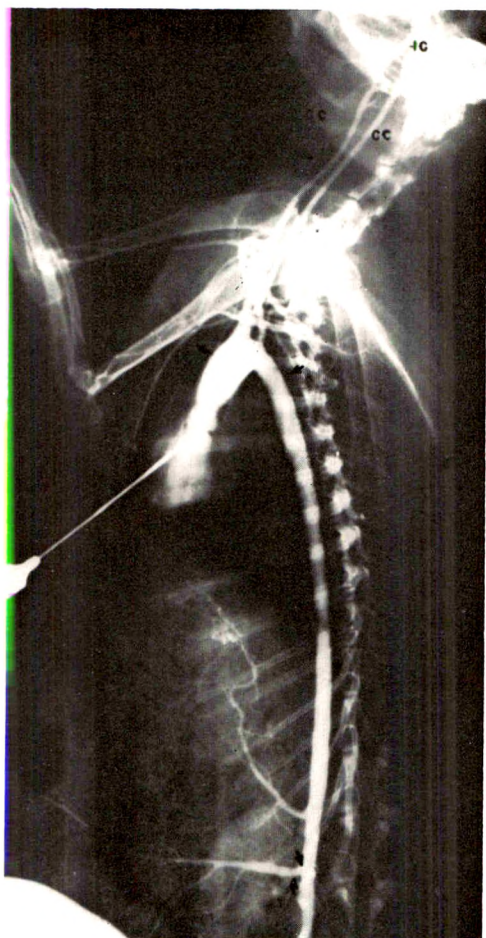


FIG. 3. Cholesterol-fed rabbit. Lateral view. Dilatation of the ascending aorta and irregular thoracic aorta; stenosis of the inferior mesenteric artery with post-stenotic dilatation. CC = common carotid; IC = internal carotid.

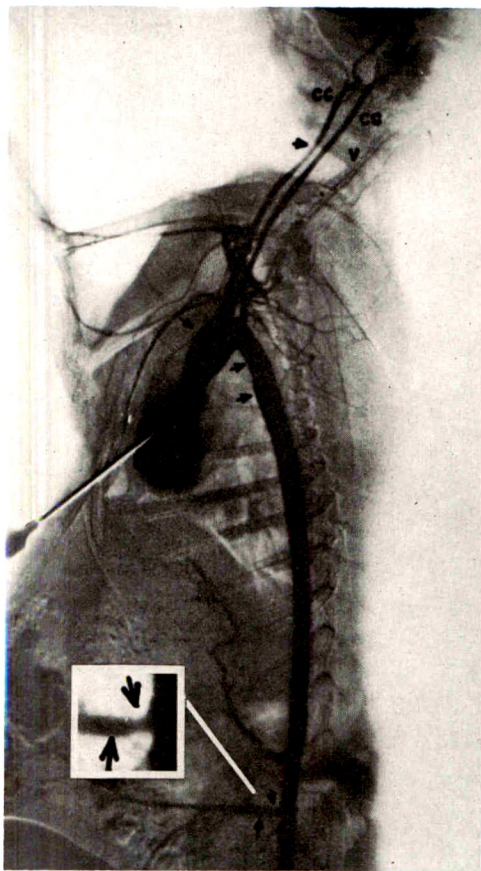


FIG. 4. Subtraction of Figure 3 with insert showing magnification ( $\times 2$ ) of the stenotic mesenteric artery. CC = common carotid.

The system was filled with contrast medium, 50 per cent diatrizoate sodium (hypaque).<sup>\*</sup> Rapid injection of 2 mm. of hypaque per kg. of body weight was made manually. The Elema-Schönander serial changer was started prior to injection of contrast medium, usually providing 4 scout films and 8 arteriographic films for subtraction studies.

If a successful puncture was made, the needle was left in place until the films were developed and examined, in case a repeat injection was necessary. The needle could be left in place as long as 30 or 40 minutes without any increase in morbidity or mortality. If the rabbit showed excessive movement during injection, the needle was removed to

<sup>\*</sup> The author wishes to thank Winthrop Laboratories for supplying diatrizoate sodium (Hypaque).



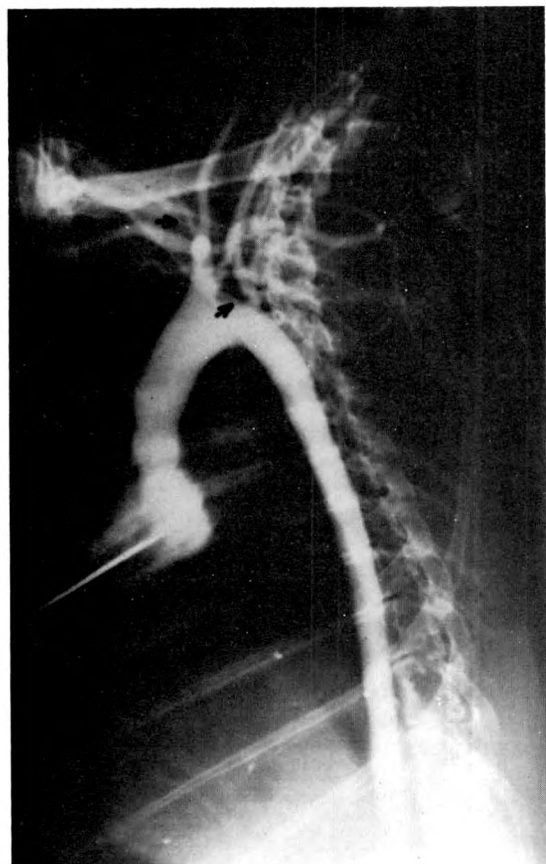


FIG. 5. Cholesterol-fed rabbit. Lateral view. Narrowing of the right common carotid artery and stenosis at the origin of the left subclavian artery (see Fig. 7, *A*, *B* and *C*).

prevent laceration of the myocardium. As a rule, the procedure was performed rapidly and the animal was sent back to quarters as soon as possible.

Prior to any injections, between injections, and at the end of all studies, the rabbit was examined by stethoscope for congestion, cardiac irregularities, and cardiac tamponade.

Anteroposterior serial roentgenograms were taken first. The animal then was turned carefully on its side for lateral serial studies. This maneuver was done quickly, easily and without complications.

Initially, pneumothorax, pneumopericardium, and hemopericardium occurred but as the technique was refined, these complications did not occur. In 2 animals, con-

trast medium was injected into the myocardium, and in 1 animal the injection was made into the pericardium. These complications were infrequent and were of minor significance. Danger of puncturing the coronary artery proved to be minimal with this procedure.

The technique can be performed by anyone familiar with the procedure of withdrawing blood from the heart.

#### POSTMORTEM STUDIES

Postmortem studies were done on all rabbits. One of the early rabbits had a needle laceration of the coronary artery with a large clot in the pericardial cavity. Several animals died after repeated ventricular punctures on the same day. In these rabbits puncture sites were noted on the surface of the left ventricle, well lateral of the



FIG. 6. Cholesterol-fed rabbit. Anteroposterior view. Marked uncoiling of the aorta and kinking of the thoracic aorta are demonstrated.

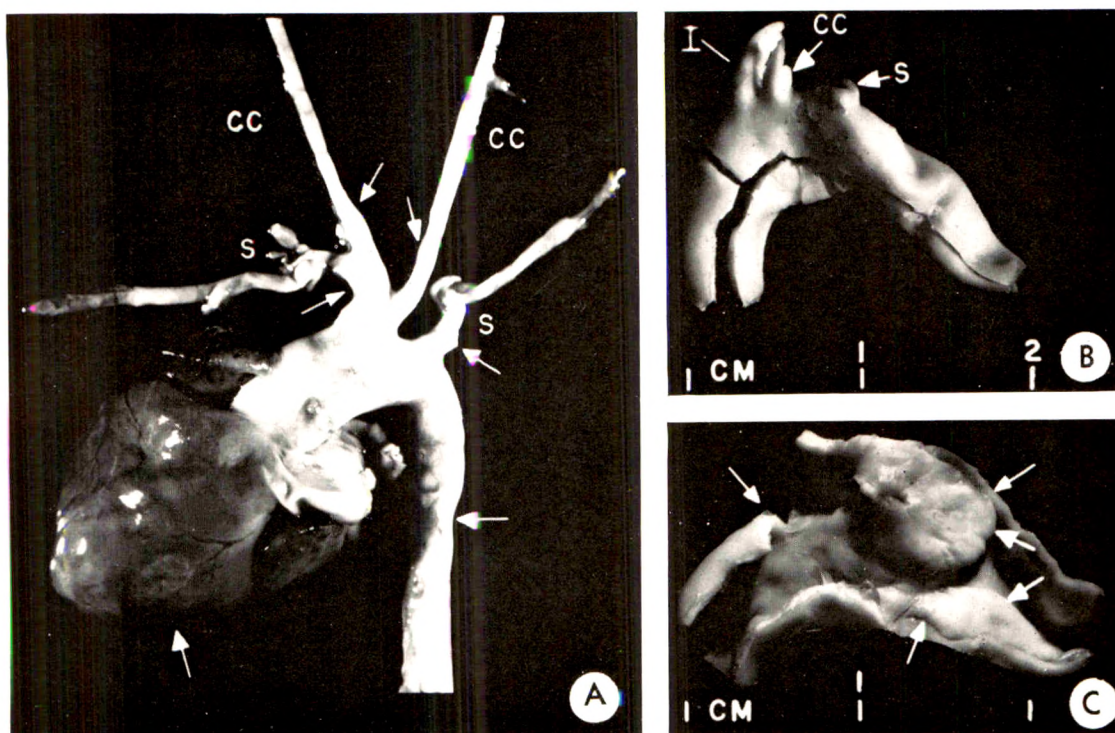


FIG. 7. (A) Cholesterol-fed rabbit. Gross specimen (see Fig. 5). Mottled white area on anterior left ventricular wall is site of previous left ventricular punctures. There is dilatation of the ascending aorta with narrowings of subclavian and common carotid arteries. (B) Adventitia removed from the aortic arch. Plaques extend from the arch into the innominate, left common carotid, and left subclavian arteries. (C) Aortic arch everted to demonstrate cobblestone appearance of the aortic intima. Vessel wall is thickened. CC = common carotid; S = subclavian; I = innominate.

coronary artery. Puncture sites could not be located with certainty when rabbits were studied weeks after the injections. In a few rabbits there were scars on the surface of the heart (Fig. 7A). (The rabbit in Figure 7, A, B and C is the same rabbit as in Figure 5.)

The heart, the aorta, and the tributary vessels, were examined totally, with the exception of the intracranial vessels and the vertebral arteries that were encased in bone. Plaques were found only in the cholesterol-fed rabbits and were concentrated, in varying degrees, in the ascending aorta, the aortic arch, the innominate artery, the common carotid and the internal carotid arteries, the subclavian and vertebral arteries, and in branches of the descending aorta (Fig. 7, A, B and C).

Severe plaques always were noted in the

autopsied vessel if arteriographic studies showed local narrowing, poststenotic dilatation, or irregular contour of a vessel (Fig. 7, A, B and C).

In all the cholesterol-fed rabbits, post-mortem studies revealed fatty livers, pale kidneys, and enlarged and whitish spleens. In some rabbits the irises contained dense radial cholesterol deposits, some had xanthomas of the extremities, and others had yellowish discoloration of the skin with severe hair loss over the anterior chest wall and abdomen.

#### DISCUSSION

Most investigators have sacrificed their animals at different stages to determine the effects of various agents or diets on prevention or regression of atherosclerosis. Left cardioventriculography provides an objec-



tive means of assessing the atherosclerotic process as it progresses in living rabbits. It should permit evaluation of anti-atherosclerotic agents or diets in reversing or holding the process, without sacrificing the animals, and without the need for catheterization or surgical procedures. At the present time, sequential studies of cholesterol-fed rabbits are being done by selective catheterization of individual vessels to determine whether or not smaller plaques can be detected earlier.<sup>1</sup>

Left cardioventriculography also provides good visualization of the arterial tree, including the cerebral and peripheral vascular systems. It can be used for the investigation of pathophysiologic states in renal, gastric or tumor circulations. Pulmonary circulation can be studied by changing the angle of the needle insertion so that it enters the right ventricle.

#### SUMMARY

A simple technique of left cardioventriculography was developed to provide a reliable method for sequential studies of atherosclerosis in the rabbit. A subtraction technique, used to remove obscuring bone

structures, enhance contrast detail and outline changes in vessels, was employed.

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## DIVERSION OF THE INFERIOR VENA CAVA INTO THE LEFT ATRIUM FOLLOWING ATRIAL SEPTAL DEFECT CLOSURE\*

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**I**NADVERTENT diversion of the venae cavae into the left atrium is an uncommon complication of atrial septal defect repair. The following cases illustrate the problems created by this type of right-to-left shunt.

### REPORT OF CASES

**CASE 1.** A 38 year old white woman was first seen in Barnes Hospital in April, 1959 with shortness of breath of 3 years' duration. The blood pressure was 115/70 mm. Hg. Cardiac auscultation revealed a Grade II harsh systolic ejection murmur in the pulmonic area. The hemoglobin was 15.5 gm. per 100 ml. Roentgenography of the chest demonstrated cardiomegaly with right ventricular enlargement and pulmonary vascular engorgement (Fig. 1A). Cardiac catheterization and angiography were performed on April 6, 1959. The catheter easily entered the left atrium from the right atrium. Right ventricular pressure was 30/0 mm. Hg. Oxygen sampling indicated a moderate left-to-right shunt at the atrial level and a left atrial cardiac angiogram revealed a large secundum-type atrial septal defect with a small inferior edge of septum. On June 10, 1959, utilizing total body hypothermia and inflow occlusion, the defect was closed with a running silk suture. The postoperative course was uneventful.

The patient was re-admitted 10 months later (April 27, 1960) because of cyanosis which, in retrospect, she had first noted 1 week after surgery. She was asymptomatic and exercise tolerance was better than before surgery. No heart murmur was detected but she had definite cyanosis of the nails and lips and early clubbing of the fingers. The electrocardiogram was normal. The hemoglobin was 18.5 gm. per cent, red blood cell count 6.01 million per cu. mm., and hematocrit 57 per cent. The chest roentgenogram demonstrated a marked interval

decrease in heart size and vascular congestion (Fig. 1B). Cardiac catheterization through the superior vena cava revealed no left-to-right shunt at the right atrial level. The catheter did not enter the inferior vena cava from the right atrium. Right ventricular pressure was 15/0 mm. Hg and a right ventricular cardiac angiogram was normal (Fig. 2). Pulmonary function tests showed an arterial oxygen saturation of 82.4 per cent at rest which rose only to 85 per cent with hyperventilation. While the patient breathed 40 per cent oxygen, the saturation was 93.1 per cent and on 100 per cent oxygen saturation it rose to 96.8 per cent. These results were considered to be compatible with a shunt of desaturated blood into the left side of the heart. Cardiac angiography performed on May 20, 1960 with injection of contrast material into the left subclavian vein failed to demonstrate any abnormal connection between the superior vena cava and the left atrium. The right atrium appeared normal (Fig. 3, A and B).

The patient was re-admitted to the hospital in July, 1961, 25 months after surgery. Marked cyanosis and clubbing were present. Cardiac catheterization was performed from the left saphenous vein and the catheter readily entered the left atrium from the inferior vena cava. Oxygen saturation was 67 per cent in the inferior vena cava, 80 per cent in the left atrium and 95 per cent in a pulmonary vein. Contrast material injected into the inferior vena cava below the diaphragm passed directly into a moderately enlarged left atrium (Fig. 4, A and B).

On October 24, 1961, 28 months after her initial surgery, re-operation was performed using the Mayo-Gibbon heart-lung machine. The inferior 1 cm. of the previously closed defect was open with the predominant flow of blood directed from the inferior vena cava into the left atrium. The original repair was taken down and reclosed with arterial silk. The patient

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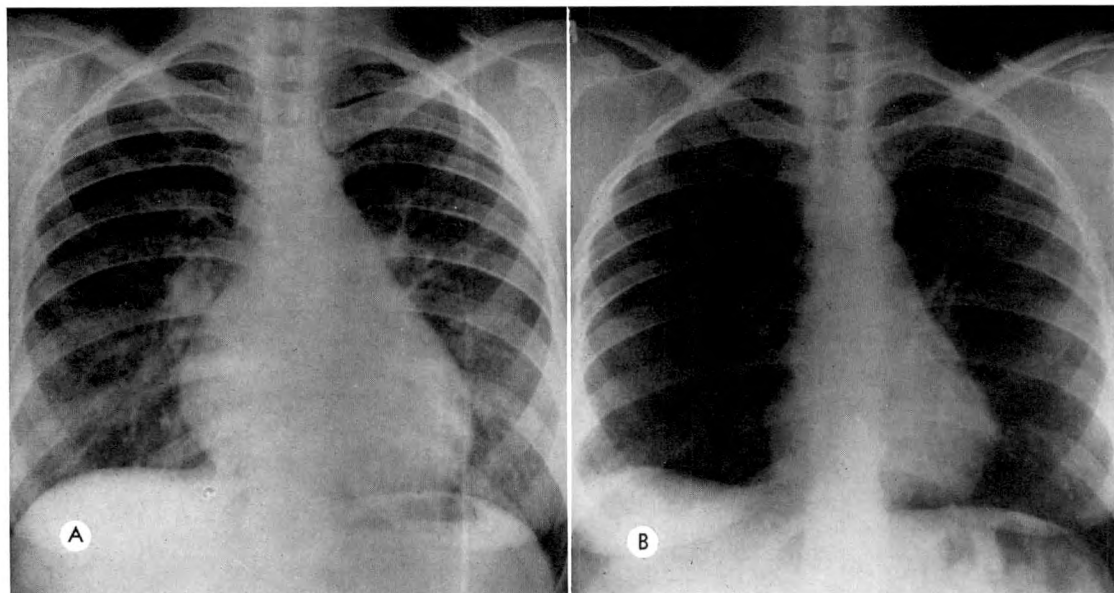


FIG. 1. Case 1. (A) Preoperative roentgenogram of the chest demonstrating right ventricle, pulmonary artery and peripheral vascular enlargement. (B) Postoperative roentgenogram 1 year following surgery. The heart and pulmonary vessels are normal.

never regained consciousness and expired 2 hours following the operation. Postmortem examination demonstrated slight cardiomegaly, a small old myocardial infarct on the lateral wall of the left ventricle, and some mild sub-endocardial hemorrhage.

CASE II. A 21 year old Negro male was admitted to Barnes Hospital in March, 1965. A heart murmur had been discovered at the age of 10 years but growth and development were normal. He participated in sports and did not limit his activity until advised to do so at the age of 16 years. He was working as a day laborer when he developed pleuritic right anterior chest pain which led to his hospital admission. Blood pressure was 135/88 mm. Hg. Cardiac examination revealed a right ventricular heave and a Grade III pulmonic systolic ejection murmur with a fixed split of the second sound. Physical examination was otherwise negative. The hemoglobin was 14.3 gm. per cent and the hematocrit 43 per cent. The electrocardiogram showed a right axis deviation and right ventricular and right atrial enlargement. The chest roentgenogram demonstrated right ventricular enlargement, prominent right ventricular outflow tract, and increased pulmonary vascularity (Fig. 5A). Cardiac catheterization and angiography confirmed the clinical diagno-

sis of atrial septal defect. Pulmonary flow was calculated as 3.7 times systemic, and the right ventricular pressure was 45/5 mm. Hg. Although the catheter entered a right pulmonary vein, a cine-angiogram suggested that the vein emptied into the left atrium.

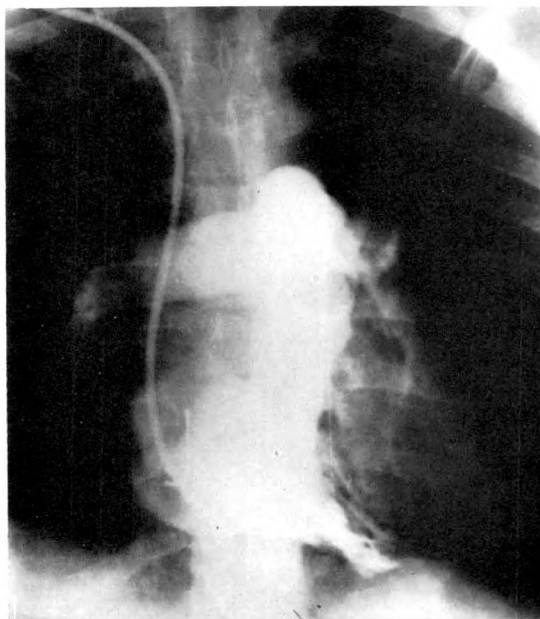


FIG. 2. Case 1. Postoperative right ventricular angiogram showing no shunt.

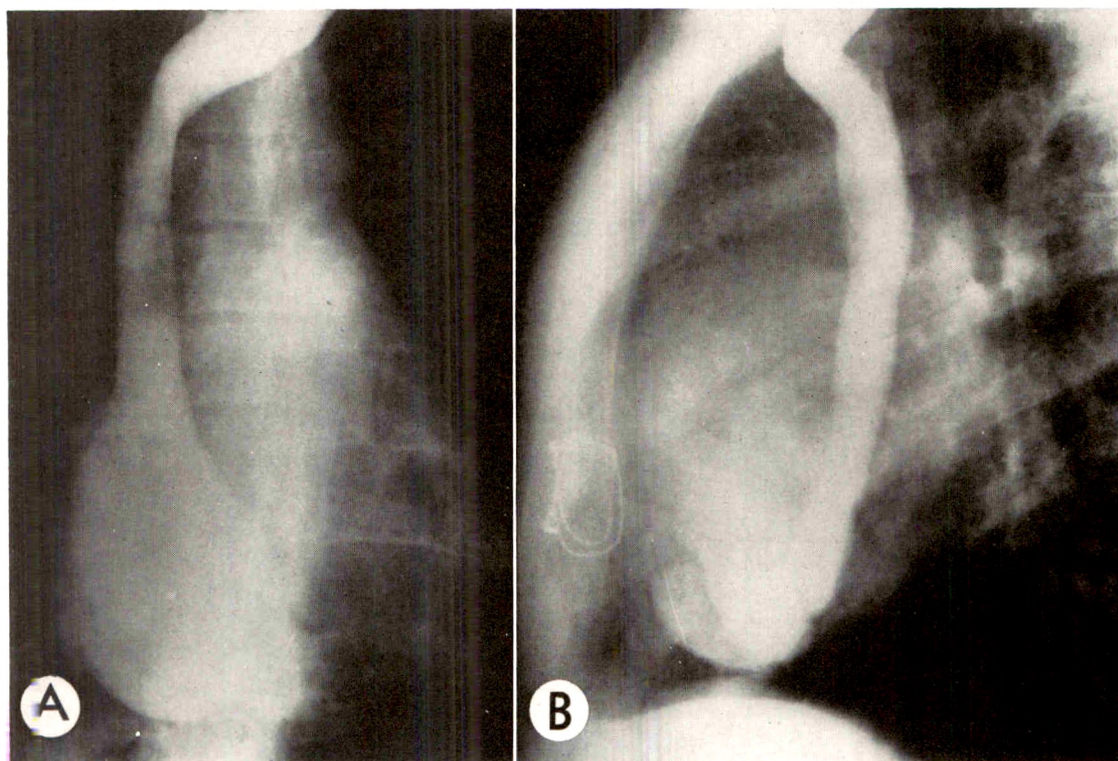


FIG. 3. Case 1. (A and B) Angiograms with injection through the left subclavian vein demonstrate a normal sized right atrium without a shunt.

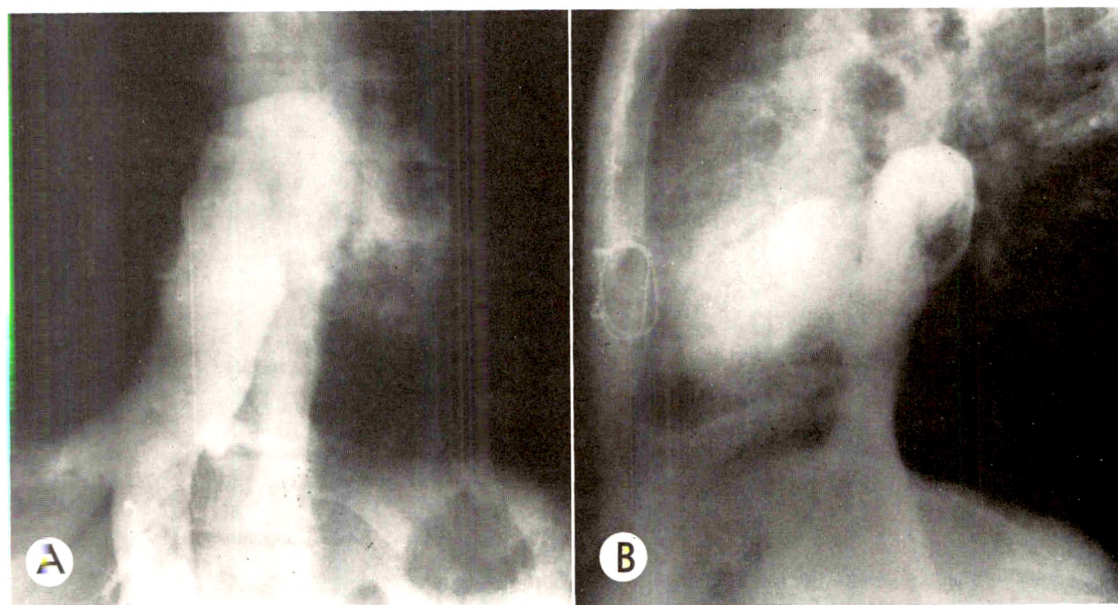


FIG. 4. Case 1. (A and B) Angiograms with injection into the inferior vena cava demonstrate prompt flow of contrast material into the left atrium and no shunt to the right.



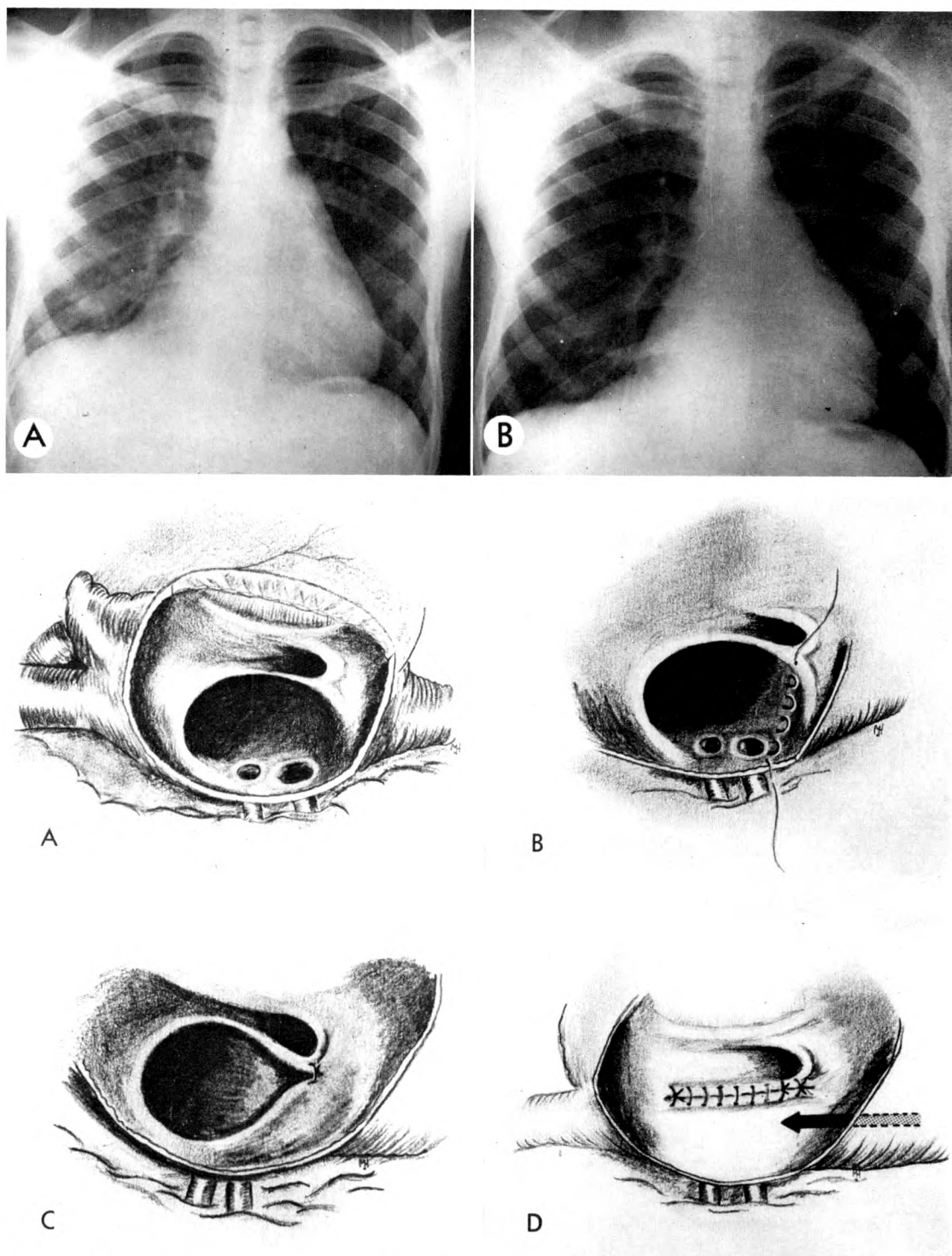


FIG. 6. Case II. Schematic representation of the surgical procedures. The septum is viewed through the opened right atrium. (A) At the first operation, the lower portion of the defect merges with the inferior vena cava. The right middle and lower lobe pulmonary veins drain into the right atrium. (B) A plication stitch is placed along the area of the inferior margin. (C) The suture is closed as a purse string. (D) The remainder of the defect is closed so the anomalous pulmonary veins are directed into the left atrium.

FIG. 5. Case II. (A) Preoperative and (B) postoperative roentgenograms demonstrate no interval change in the appearance of the enlarged heart following repair of the atrial septal defect.

Thoracotomy was performed on April 21, 1965 using a disposable bag oxygenator. The superior and inferior venae cavae were cannulated through the right atrium. A large ostium secundum defect was found very low in the septum. There was no inferior rim so that the lower portion of the defect merged with the inferior vena cava. The right middle and lower lobe pulmonary veins drained anomalously into the right atrium (Fig. 6A). The defect was closed with interrupted silk sutures placed so that pulmonary veins were redirected to the left atrium. An inferior rim was created by a plication stitch which ran from the margins of the defect along the left lateral wall of the inferior vena cava, thus assuring drainage of the inferior vena cava into the right atrium (Fig. 6, B, C and D). He was re-explored 24 hours later for ligation of a bleeding internal mammary vein. Postoperatively, the patient's mucous membranes and nail beds appeared cyanotic and arterial oxygen saturations were low. This was thought to be due to pulmonary congestion. On the day of discharge (May 4, 1965), arterial oxygen saturation was 79 per cent. Repeat arterial oxygen saturation was 89 per cent on May 24, 1965. Ventilatory studies were normal.

The patient was re-hospitalized on July 13, 1965. Cyanosis persisted and the hemoglobin had risen to 17.3 gm. per cent and the hematocrit to 53 per cent. Roentgenography of the chest showed no change in heart size or pulmonary vessels (Fig. 5B). A gray Ödman catheter inserted percutaneously into the right femoral vein by the Seldinger technique passed readily into the left atrium. Angiograms demonstrated a normal left atrium and no left-to-right shunt (Fig. 7, A and B). A second injection was made into the inferior vena cava just below the diaphragm. In the right posterior oblique projection, the stream of contrast material from the inferior vena cava was clearly seen to divide, with some entering each atrium (Fig. 8A). The left posterior oblique projection showed narrowing of the inferior vena cava at the site of the septal repair (Fig. 8B).

Re-exploration on September 16, 1965 showed that the reefing stitch in the inferior aspect of

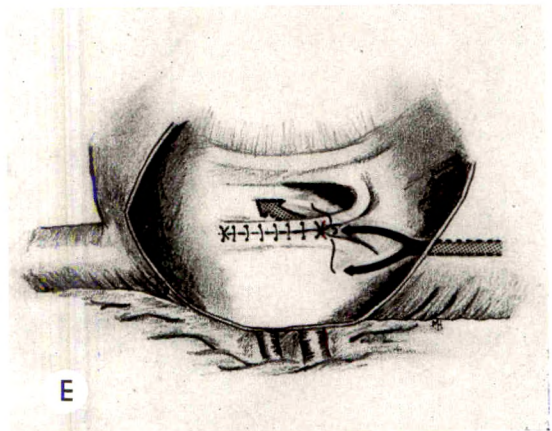


FIG. 6. (E) Reoperation for correction of the right-to-left shunt. The plication suture had opened, dividing the flow from the inferior vena cava into both atria.

the septal defect had pulled out while the remainder of the repair had remained intact (Fig. 6E). This allowed the atrial septum to override the vena cava, dividing the flow into two separate streams with approximately half going to each atrium. The atrial septum was re-opened and a teflon patch was sewn in place. Postoperatively, the arterial oxygen saturation was 100 per cent. Cyanosis was absent. However, the patient experienced respiratory difficulty and showed progressive mental deterioration. He died 24 hours after surgery. Autopsy permission was not obtained. Death was attributed to cerebral damage secondary to poor perfusion or air embolism.

#### DISCUSSION

Diversion of the inferior or superior venae cavae into the left atrium is an unusual and infrequently discussed complication to atrial septal defect closure.<sup>1-7</sup> The limited dissemination of knowledge concerning this complication led to a delay in its recognition in Case I. In Case II, the possibility of connection between the inferior vena cava and left atrium was immediately recognized and the diagnosis was



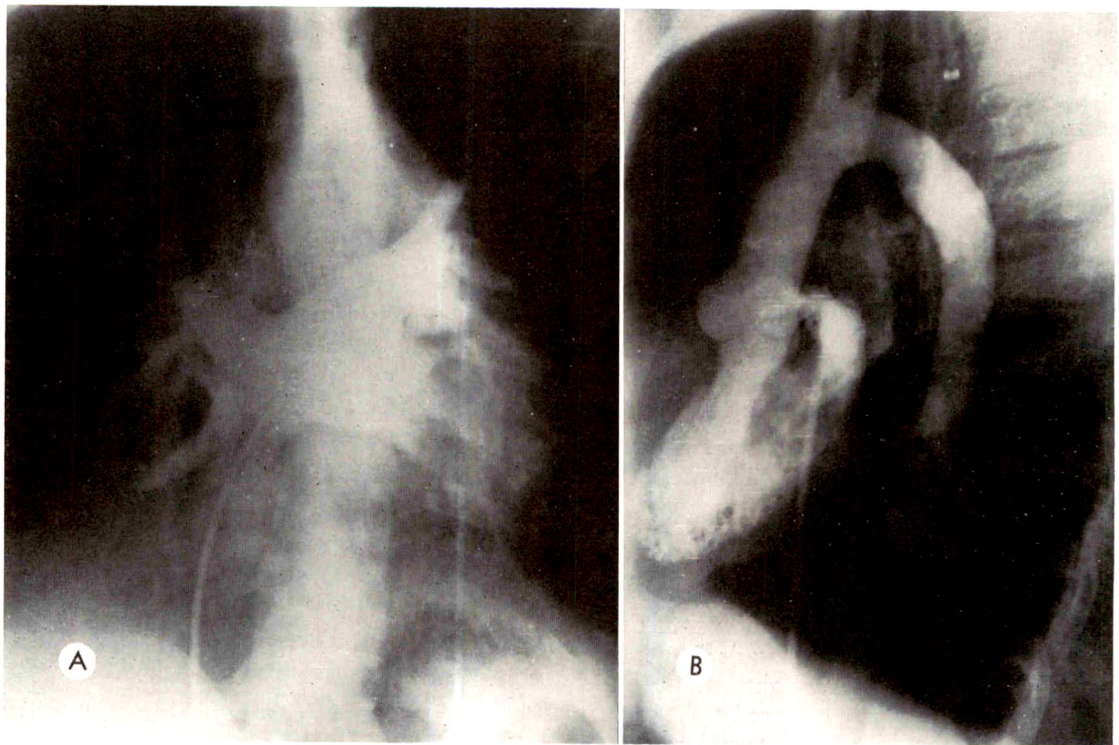


FIG. 7. Case II. (A and B) Angiograms with injection into the left atrium with the catheter passed from the inferior vena cava demonstrate no shunt of contrast material into the right atrium. The upper portion of the atrial septum is intact.

confirmed when the patient agreed to re-catheterization. The 2 patients presented here are the only ones with this complication in the series of over 200 atrial septal defect repairs during the past 9 years.

The incidence of vena caval diversion is probably higher in cases in which only hypothermia and inflow occlusion are used due to the time limitations imposed. Cardiopulmonary bypass allows ample time to assess and repair the defect but has not led to complete elimination of this complication. Common factors in the reported cases are a large secundum-type or sinus venosus-type defect which blends with either the inferior or the superior vena cava, and anomalous pulmonary venous return into the right atrium.<sup>1,6,7</sup> Anomalous pulmonary venous drainage requires placement of the sutures to the right of the vessel orifice to divert the flow into the left atrium. In doing this the right atrium may be distorted so that the superior or inferior vena

caval blood is also diverted into the left atrium along with the flow from the anomalous pulmonary vessels. Indeed, in 1 instance of repair of a sinus venosus defect, distortion resulted in occlusion of the superior vena cava. Since bypass had already been discontinued, emergency repair was made by anastomosis of the superior vena cava to the right atrial appendage, resulting in a successful outcome.<sup>7</sup> The use of a graft has been advocated by some authors as a means of preventing the distortion resulting from repair of a large defect.<sup>6</sup>

An enlarged eustachian valve of the inferior vena cava may be mistaken for the inferior rim of the atrial defect and used in the repair.<sup>5</sup> This diverts flow from the inferior vena cava to the left atrium. A catheter placed through the right atrium to the inferior or superior vena cava as a guide does not necessarily assure proper placement of the sutures. If utmost care is not



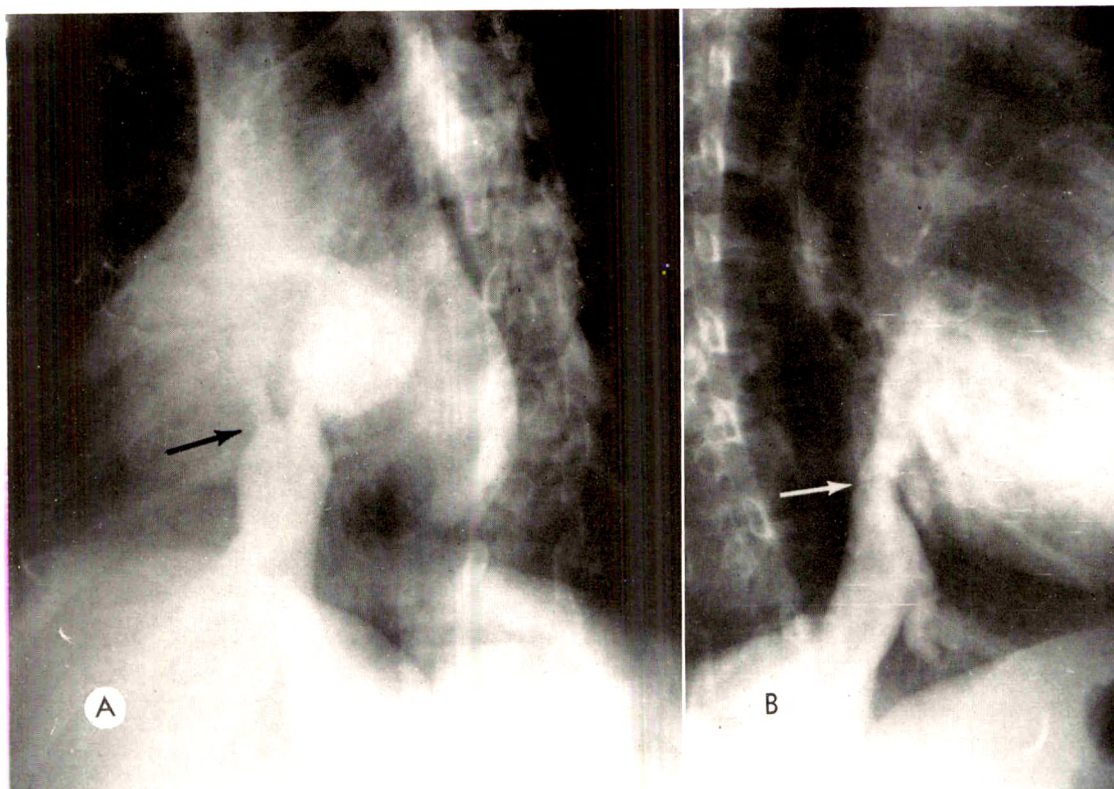


FIG. 8. Case II. (A) Right posterior oblique and (B) left posterior oblique projections following injection into the inferior vena cava. The flow from the inferior vena cava is directed into both atria at the inferior aspect of the septum (arrow in A). The inferior vena cava is narrowed at its entrance into the atria (arrow in B).

taken to close completely the inferior angle behind the catheter, sufficient distortion may occur so that a part of the inferior vena cava is displaced into the left atrium after removal of the cannula. Blood flow into the right atrium will obscure the true anatomic situation.

The appearance of cyanosis following an atrial septal defect repair in a patient who is otherwise doing well should immediately suggest the possibility of vena caval diversion into the left atrium. Cyanosis may appear while the patient is still under the anesthesia or several months or years later. If the shunt is small it may be manifest only during exercise or a bout of respiratory infection.<sup>6</sup> The chest roentgenogram in the postoperative period may show a return of the heart and pulmonary vessels to normal despite the right-to-left shunt.<sup>3,7</sup> Another cause of cyanosis is suggested in patients

with severe pulmonary vascular disease or postoperative pulmonary complications. In the latter groups, severe illness would be present and the cause of cyanosis should be evident.

Diagnostic work-up to establish the nature of the complications should be guided by the location of the repaired defect. A defect near the inferior vena cava should be investigated by catheterization from the inferior vena cava with injection of contrast material into the cava itself. The oblique projection best demonstrates the relationship of the vena cava to the atrial septal defect. In the case of high defects of the sinus venosus type, catheterization should be performed through the superior vena cava with injection into the cava. Usually, the catheter will readily enter the left atrium as it is advanced further. Because there is usually a small defect re-



maining at the entrance of the vena cava into the atrium, the catheter may occasionally pass into the right atrium. However, the vena caval injection will demonstrate the true anatomic situation. An injection of contrast material into the left atrium will allow assessment of the integrity of the remainder of the atrial septum.

Diversion of a cava into the left atrium would appear to warrant re-operation to correct the complication. The right-to-left shunt causes secondary polycythemia and presents a significant risk of paradoxical embolism. Re-operation has been performed during the initial anesthesia as soon as arterial desaturation was observed and as late as 4 years after the initial operation with good results.<sup>1,6,7</sup>

#### SUMMARY

Two patients are presented in whom repair of an atrial septal defect of the secundum type resulted in a diversion of the inferior vena cava to the left atrium. Cyanosis or arterial oxygen desaturation following surgery was the first indication of this complication. Diagnostic work-up to establish the nature of the complication should be guided by the location of the repaired defect.

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## OBSERVATIONS ON PULMONARY EMBOLISM AND THE PULMONARY ANGIOGRAM\*

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**P**ULMONARY embolism, the most frequent acute respiratory disease encountered in a general hospital,<sup>16,18</sup> poses a difficult diagnostic problem to the radiologist. This challenge assumes even greater importance when sequelae, both acute and chronic, are potentially life threatening. Unfortunately, many embolic episodes produce neither symptoms<sup>8</sup> nor detectable change on the chest roentgenogram.<sup>11</sup> In these instances, suspicion is the radiologist's best guide. A suggestive history, supported by nonspecific alterations of the electrocardiogram, blood chemistries and chest roentgenogram, may lead to a proper diagnosis. This may be sufficient for conventional therapy, but greater certainty is often required.

Our ability to diagnose and evaluate pulmonary emboli has been improved and extended by pulmonary angiography and by hemodynamic data obtained from right heart catheterization. These have provided a more quantitative measure of the obstructive process, greater reliability for diagnostic purposes and have better enabled us to determine the need for inferior vena cava ligation, or pulmonary artery surgery. In many instances, the additional studies have been necessary in order to exclude pulmonary embolism as a diagnostic consideration.

Since most patients with pulmonary emboli do not die, clinical pathologic correlation is difficult. The following study on patients with suspected pulmonary emboli, proved by alterations in their hemodynamic data and by the pulmonary angiogram, has provided a better basis for an analysis of the patient with pulmonary embolism.

### METHODS AND MATERIALS

A total of 58 patients were referred to the authors from February, 1964 to December, 1965, with a tentative diagnosis of pulmonary embolism. In addition to routine evaluation, right heart catheterization was performed, followed by pulmonary angiography via a catheter in the pulmonary artery. This sequence was maintained in order to ensure that pressure alterations were not produced by the contrast material. The main pulmonary artery pressure was recorded of all patients. As a more sensitive index of occlusive vascular disease, pulmonary hemodynamic data were obtained following unilateral pulmonary artery balloon occlusion<sup>2,3,6</sup> in 19 patients and are being reported separately.<sup>10</sup>

Pulmonary angiography was performed with the patient in the supine anteroposterior position. Where necessary and, when feasible, oblique projections and selective right or left pulmonary artery injections were utilized for further evaluation. Using a Zimmerman pump, 50–60 cc. of 75 per cent hypaque was injected and films serially exposed at 2 per second, using a Sanchez-Perez or Schönander film changer.

Eight patients had repeat hemodynamic and angiographic studies. These were done to determine if repeat embolic episodes had occurred during anticoagulant therapy, or followed inferior vena cava ligation; or, to determine the evolution of previously demonstrated emboli.

The history, physical examination, laboratory values, electrocardiogram, chest roentgenogram, right heart catheterization data and angiogram were reviewed on all patients. If either the history, pulmonary

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TABLE I  
AGE DISTRIBUTION—EMBOLIC GROUP

Age	No. of Cases
10-19.....	1
20-29.....	3
30-39.....	9
40-49.....	9
50-59.....	6
60-69.....	8
70-79.....	2

angiogram, pulmonary artery pressure, or appropriate surgical or autopsy findings gave evidence of a pulmonary embolus, the patient was included in the embolic group. On this basis, it was determined that 38 patients had one or more pulmonary embolic episodes. The findings in this group were further evaluated, especially with reference to the chest roentgenogram and the pulmonary angiogram. The normal angiogram, as recorded for the nonembolic group and an additional 30 cases (studies for reasons other than pulmonary emboli), was compared with that of the embolic group.

RESULTS

On the basis of the criteria described above, 38 patients had either single or multiple pulmonary emboli; 20 had no evidence of embolic disease.

CLINICAL FINDINGS

In the embolic group, there were 21 females and 17 males, with an age range from 15 to 78 years (Table I). Many of the elements conducive to embolic episodes were recorded: trauma (9), phlebitis (18), post-operative (6), cardiac failure (4), mitral stenosis (2), post partum (2) and polycythemia vera (1). No evident source for emboli could be found in 10 patients.

Both acute and chronic cardiorespiratory complaints, as well as nonspecific systemic disturbances prompted referral (Table II). Acute chest pain was the most frequent presenting symptom, and, in this small series, when associated with hemoptysis,

was due to an embolus. No other finding was as helpful.

CHEST ROENTGENOGRAPHY

Eight patients with pulmonary emboli had a normal chest roentgenogram; the angiogram confirmed the diagnosis in 4. Another group of 4 patients had either a normal chest roentgenogram at the time of the acute embolic episode but later developed parenchymal and/or pleuritic densities; or had a density which appeared transiently and then cleared rapidly. Four patients without lateralizing chest symptoms had bilateral emboli. Six patients had unilateral chest symptoms and unilateral changes on chest roentgenograms with pulmonary angiograms indicative of bilateral disease. Ten patients had unilateral chest symptoms, bilateral changes on chest roentgenograms, and emboli demonstrated bilaterally by the angiogram.

TABLE II  
PRESENTING SYMPTOMS

	Embolic Group	Non-embolic Group
Acute Chest Pain.....	31	12
with hemoptysis.....	11	
without hemoptysis....	20	12
with fever.....	6	
with dyspnea.....	6	0
with tachycardia.....	4	
with congestive heart failure.....	2	0
Right Upper Quadrant Pain....	2	
with dyspnea.....	1	
with hemoptysis.....	2	
Without Associated Pain.....	5	8
congestive heart failure and dyspnea.....	1	4
fever.....	1	3
tachycardia.....	1	1
hypotension.....	2	1
dyspnea.....	3	3

The majority of patients had alterations of the lung bases (Table III), with elements of the classic triad of pleuritis, atelectatic plaques and diaphragmatic elevation, occurring either singly or in combination. These were combined with densities in additional portions of the lung in 7 patients. Mid and upper lobe pneumonia-like densities were the sole roentgen change in 2 patients.

#### HEMODYNAMIC STUDIES

Twenty-four of the 38 patients with thromboembolism had resting pulmonary hypertension. Seventeen of the 38 patients in the embolic group were further examined by unilateral pulmonary artery balloon occlusion. Eight patients had a normal pulmonary artery pressure at rest; following balloon occlusion, the pulmonary artery pressure increased in 6. The other 9 patients had resting pulmonary hypertension; following unilateral occlusion, the pulmonary artery pressure remained unchanged in 5, but further increased in the remaining 4.

#### PULMONARY ANGIOGRAPHY

Emboli were demonstrated by angiography in 32 of the 38 patients. This includes 21 of the 24 patients with resting pulmonary hypertension and 11 of the patients with a normal resting pulmonary artery pressure. As indicated above, 16 patients had bilateral embolic disease in spite of unilateral symptomatology and/or unilateral plain film alteration. Embolic changes were also demonstrated by angiography in: (a) 8 of the 9 patients with pulmonary hypertension who had the unilateral pulmonary artery balloon occlusion; (b) 4 out of the 6 patients with normal pulmonary artery pressure at rest and with the positive pressure response to balloon occlusion; and (c) the 2 patients with normal pulmonary artery pressure and a normal response to balloon occlusion.

The normal pulmonary angiogram, as recorded serially, shows smooth, gradually tapering arteries which fill uniformly throughout both lung fields, and which

TABLE III  
FINDINGS ON CHEST ROENTGENOGRAMS

	Embolic Group	Non-embolic Group
Abnormality of Pulmonary		
Artery.....	4	—
Westermarck Sign.....	2	—
Pneumonia.....	9	2
Basilar Lung Changes.....	25	9
Plaque.....	10	5
Pleuritis.....	17	6
Elevation of diaphragm.....	12	2
Infarct shadow.....	3	1
Negative Chest.....	8	9

simultaneously opacify the peripheral vessels. There is a similar, uniform opacification of the capillary phase, as well as venous return. While the segmental anatomy of the lung, as it appears on the angiogram, has been recently clarified by Jefferson,<sup>17</sup> precise anatomic evaluation similar to bronchographic analysis did not help us to identify emboli.

Emboli present characteristic features in the pulmonary angiogram, depending upon their age, size, number, and location.<sup>19</sup> The following changes were observed in our study: Larger arteries and their segmental branches, when completely obstructed, showed an abrupt cut-off of the contrast filled vessels (Fig. 1, *A-E*). The margins were concave, squared-off or, rarely, convex-forward. When complete obstruction had not occurred, a filling defect was seen (Fig. 2, *A-D*), lobular or wormy in configuration, or one which conformed to the contour of the involved vessel. Arterial stasis, noticeable during the capillary and venous stages (Fig. 3, *A* and *B*) resulted from (a) complete obstruction of a larger vessel, (b) trapping of contrast material around or within a clot, or (c) microembolic obstruction to peripheral flow.

Localized segmental avascularity and absent, diminished, or delayed segmental venous return were occasionally observed and depended upon the number and size of



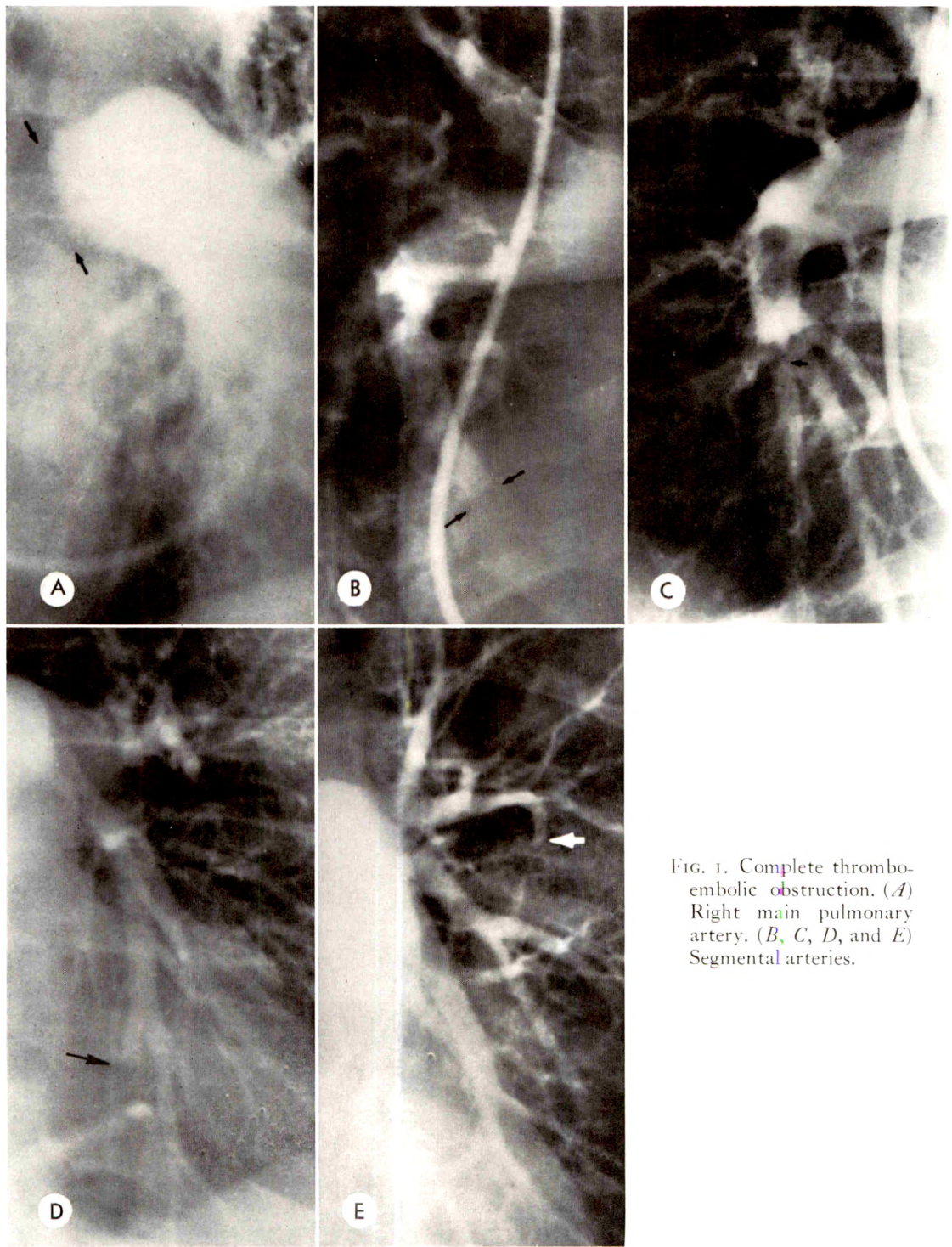


FIG. 1. Complete thromboembolic obstruction. (A) Right main pulmonary artery. (B, C, D, and E) Segmental arteries.

vessels affected, as well as the available collateral circulation. Abnormalities in the venous drainage, without associated arte-

rial or capillary change, did not establish the diagnosis in any single case. For the smaller vessels, film resolution



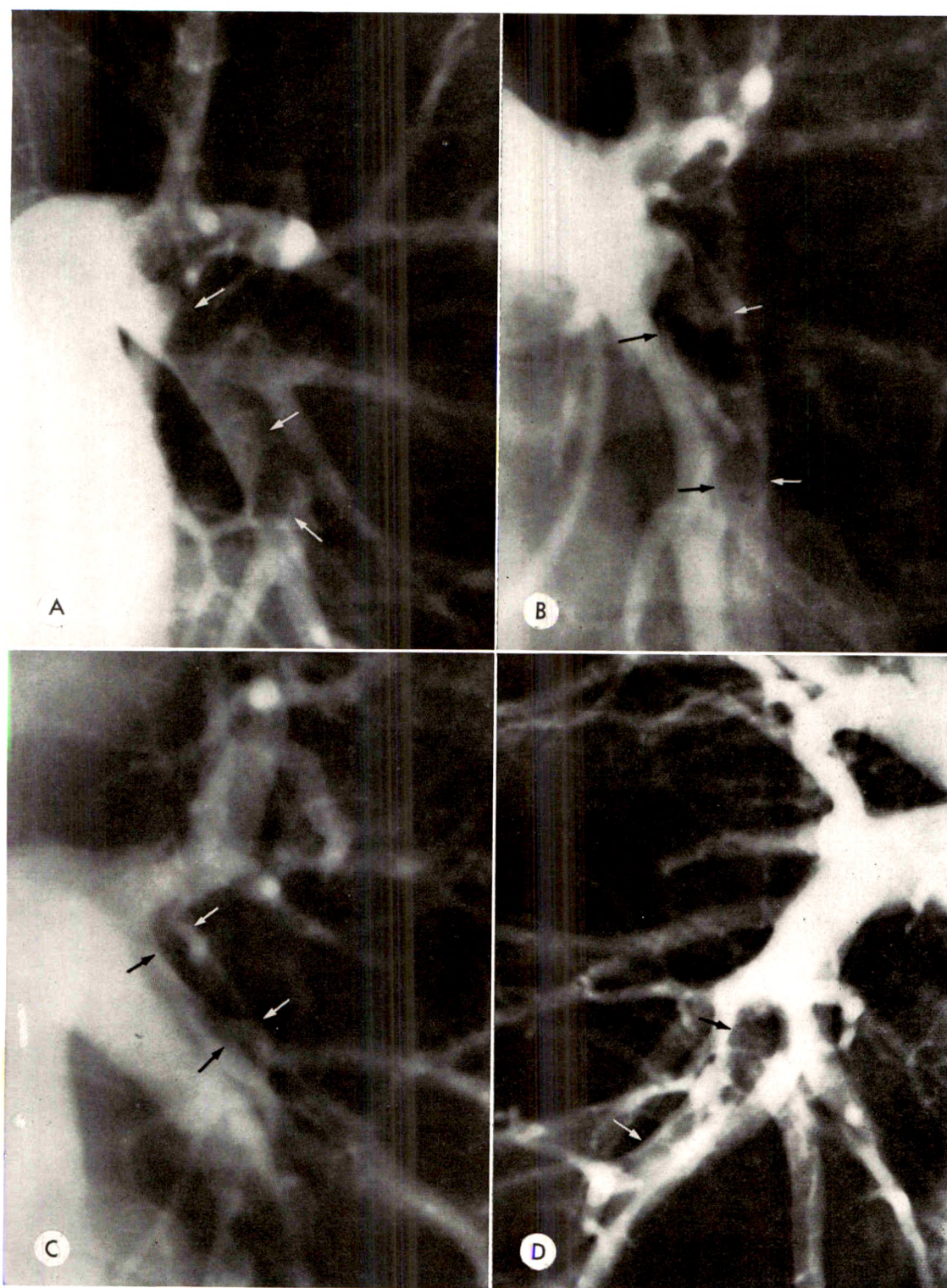


FIG. 2. Incomplete thromboembolic obstruction. (A, B, C, and D) Filling defects within lower lobe arteries.



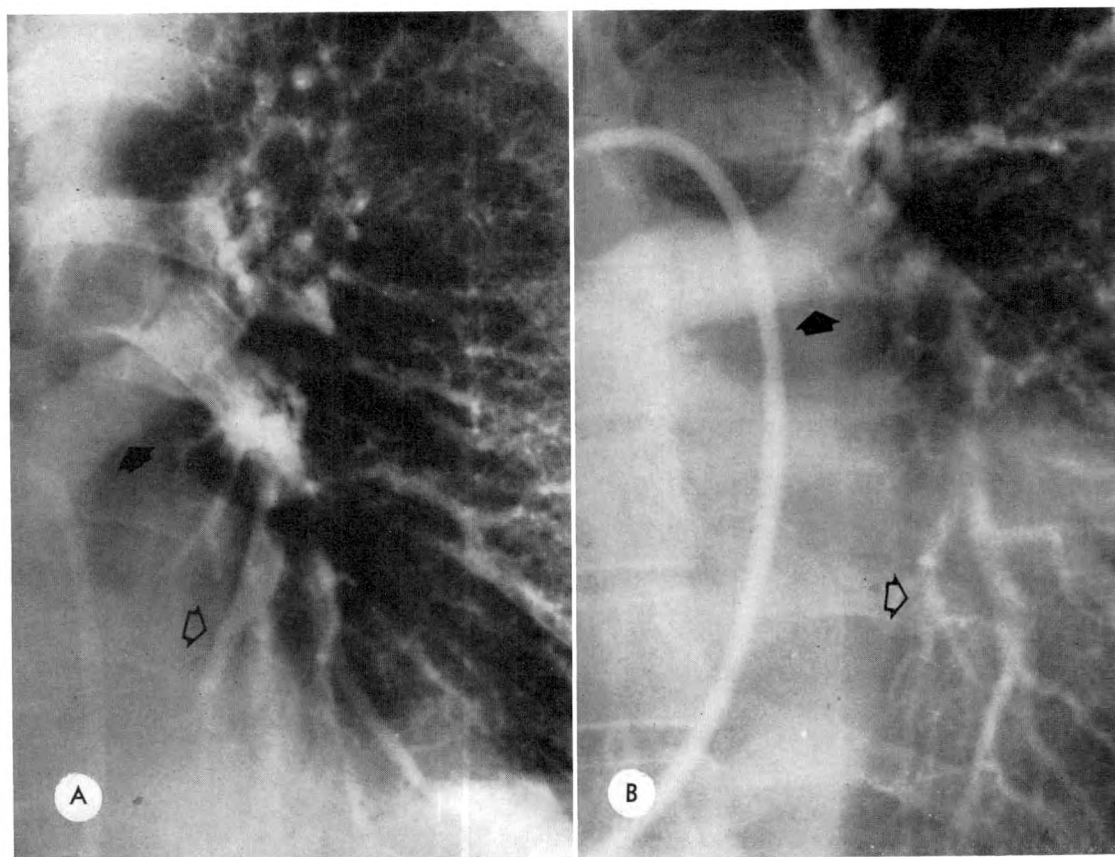


FIG. 3. (*A* and *B*) Arterial stasis. Lower lobe emboli. Persistence of contrast material within arteries during venous phase.  $\rightarrow$  = pulmonary vein;  $\Rightarrow$  = segmental arteries.

and contrast delineation were limiting factors in the diagnosis of some patients. Although not directly identified, small emboli produced segmental changes characterized by: (1) plump, segmental arteries supplying the affected area, wavy in contour, with apparent shortening; (2) relatively sparse, segmental branches; and (3) tortuous, shrunken and horizontal terminal branches, which were also diminished in number. In the extreme form, this appearance has been likened to a "pruned tree." Depending upon the degree of involvement, however, one may see all of the above changes or little more than tortuosity of the distal branches (Fig. 4, *A*, *B* and *C*; and 5, *A-D*). The small vessel changes were most frequently noted in those patients who had other larger vessel proof of embolic disease. The peripheral involvement, however, was

the only abnormality in 9 patients and was noted especially in those where several days elapsed between the clinical event and the performance of angiography.

#### DISCUSSION

##### CLINICAL DATA

The usual and customary signs and symptoms of embolic disease, both acute and chronic, undoubtedly played a role in selecting patients for this series. These were not sensitive enough to detect most emboli as was shown by the presence of emboli and the absence of localizing chest symptoms and roentgen changes. An additional 16 patients had bilateral emboli in spite of unilateral symptoms.

As stated previously, the group with chest pain and hemoptysis had emboli. Without hemoptysis, however, acute right

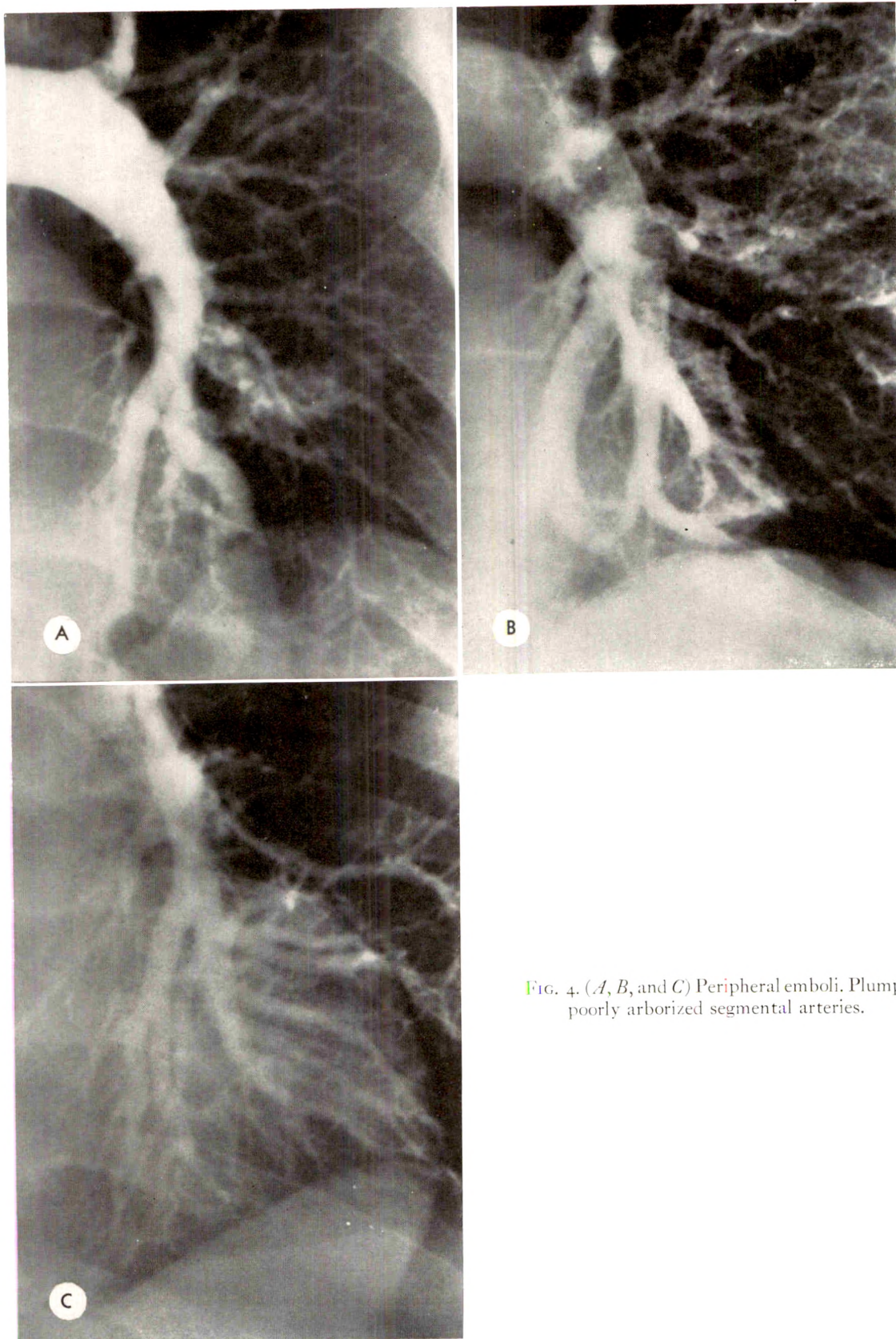


FIG. 4. (*A*, *B*, and *C*) Peripheral emboli. Plump, poorly arborized segmental arteries.



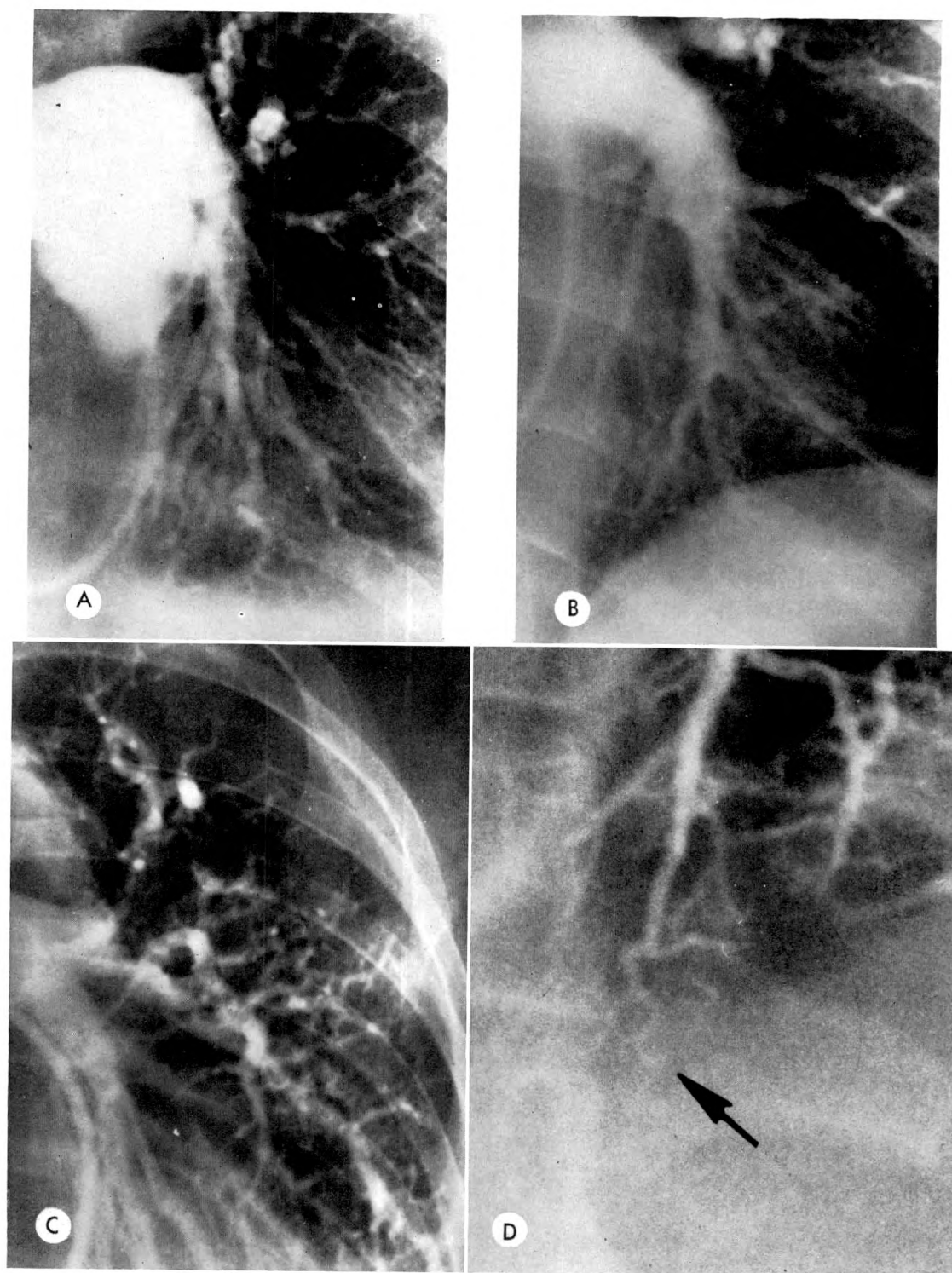


FIG. 5. Peripheral emboli. (A, B, C, and D) Tortuous segmental and/or distal arterial branches.

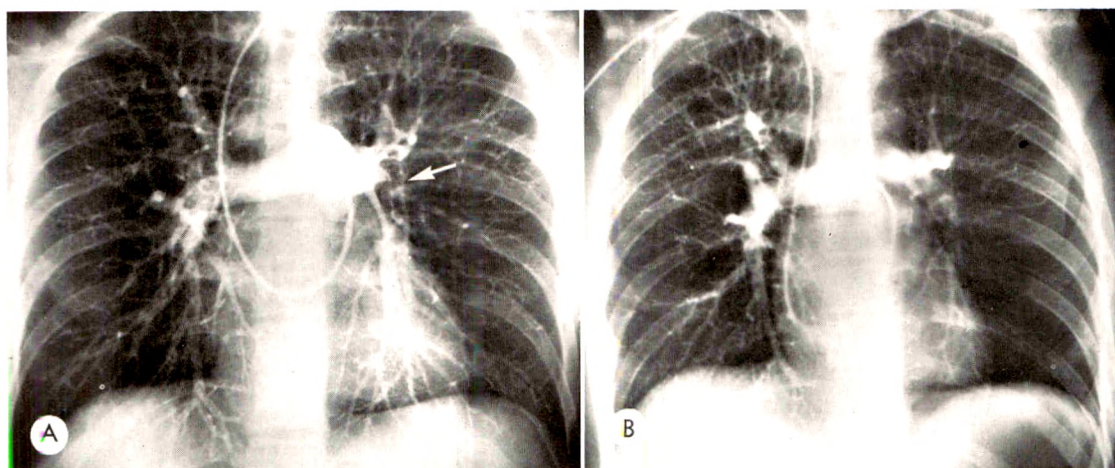


FIG. 6. (A) Asymptomatic, nonobstructing thrombus of left main pulmonary artery ( $\rightarrow$ ). (B) Two days post-inferior vena cava ligation. Distal movement of clot, obstructed left lower lobe vessels and chest pain.

lower chest pain was often a confusing symptom and was infradiaphragmatic in origin in 5 patients, due to abnormalities of the liver (hepatitis—3, cholecystitis—1, metastatic carcinoma—1). Conversely, right upper quadrant pain and a diagnosis of acute cholecystitis were entertained in 1 patient who later proved to have multiple pulmonary emboli.

More chronic chest symptoms, such as unexplained congestive heart failure or asthma, were another basis for referral. One patient in this series had frequent unexplained episodes of congestive failure, which responded poorly to conventional medical therapy. The pulmonary artery pressure was 65/25 (35) mm., and the angiogram showed obstructions of multiple branch arteries rather than the diffuse small vessel change described for patients having chronic obliterative thromboembolism.<sup>7</sup>

A smaller number of patients had no specific chest symptoms—presenting as unexplained fever or periodic or sustained tachycardia. Other patients had malaise, vague aching or episodes of tachypnea prior to their more classic presentation of acute pleuritic pain with or without hemoptysis. Clinical symptoms appeared to be more frequent when associated with obstruction to middle sized and smaller pe-

ripheral arteries. A lag in time between the embolic episode and the appearance of the infarct shadow on the chest roentgenogram has been stressed by Fleischner.<sup>12</sup> We believe that a similar lag in clinical symptoms in some patients is related to fragmentation of an already existing central thrombus, which later extends and involves a pleural surface. As an example, an inferior vena cava ligation was performed on a 30 year old Negro female whose presenting symptom was severe right chest pain. The angiogram showed small avascular segments at the right lung base and a large asymptomatic nonobstructing thrombus within the left main pulmonary artery (Fig. 6A). A repeat pulmonary angiogram was made on the second postoperative day because of left chest pain. This demonstrated that the left main pulmonary artery thrombus had moved distally and obstructed the more peripheral vessels (Fig. 6B).

#### THE CHEST ROENTGENOGRAM

Although the chest roentgenogram improved the detection of the embolic event, 8 patients had normal chest roentgenograms, 4 had transient parenchymal densities, and 6 patients had unilateral roentgenographic changes in spite of proved bilateral disease. Too great a reliability,



therefore, cannot be placed on the chest roentgenogram, since this study is often not sensitive enough and the change too transient for adequate demonstration of the presence and extent of the embolic process.

Plain roentgenograms which demonstrated a classic infarct shadow were the exception. The possibility of an embolus was frequently suggested if pleural reactions, atelectatic plaques, or alterations of the diaphragm were present. However, these are not specific changes and can be produced by a multitude of other supra- and infradiaphragmatic causes. In the absence of basilar involvement there was a greater error in diagnosis. An inflammatory process was often the initial impression. This was because the infarct shadow above the lung bases looked like a patchy pneumonic area, sometimes small and transient, simulating an artefact. Though a fissural

boundary was apparent on a few of these densities, there was no associated pleuritis. The angiogram frequently demonstrated a segmental artery occlusion leading to the infarct shadow.

Enlargement of the pulmonary artery at either hilus signaled pulmonary thromboembolism in 4 patients. In each instance, the dilated artery contained clot as proved by the angiogram. Serial chest roentgenograms and repeat angiograms showed that the return to normal arterial width was accompanied by dissolution or peripheral movement of the clot.

Westermark's sign<sup>21</sup> as an initial indication of the embolus was used in 2 cases. These had large main pulmonary artery thrombi. The disparity in lung vascularity is easier to diagnose in the mid lung areas—the natural areas of comparison. Unfortunately, most emboli involve lower lobes and

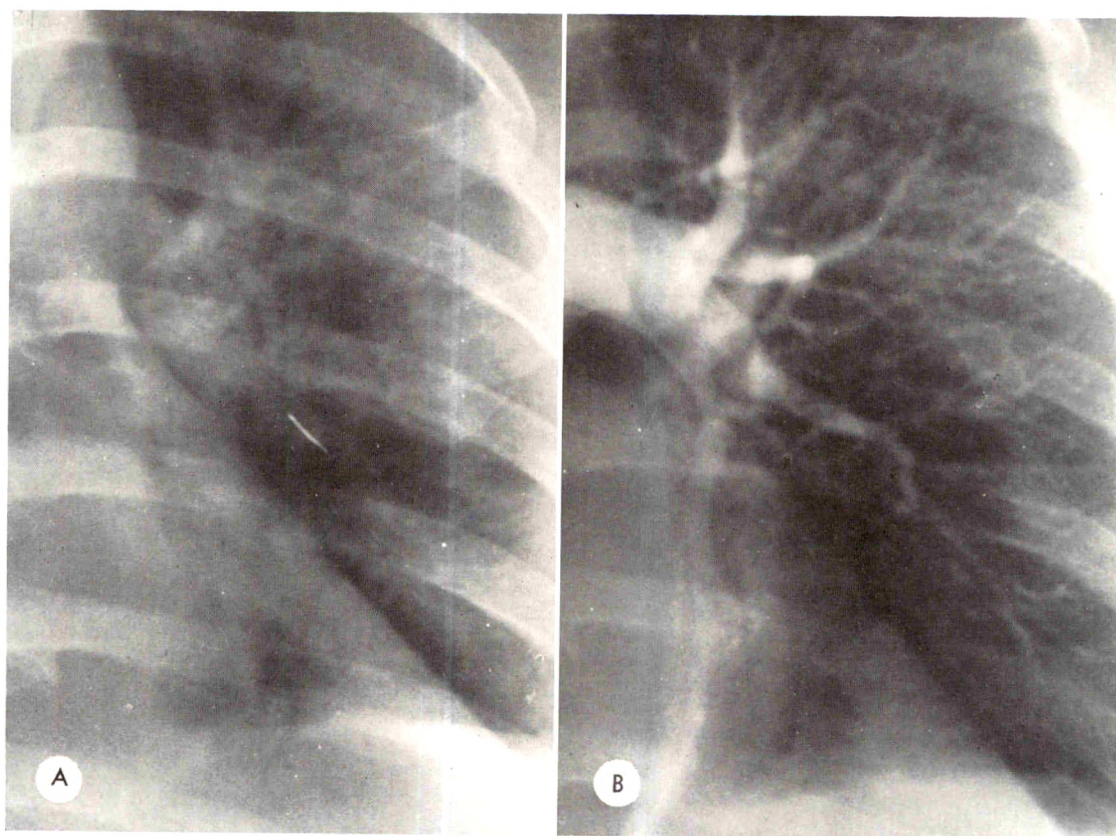


FIG. 7. Hypovascular lower lobe. (A) Overpenetrated roentgenogram prior to injection of contrast material. (B) Corresponding arteriogram.



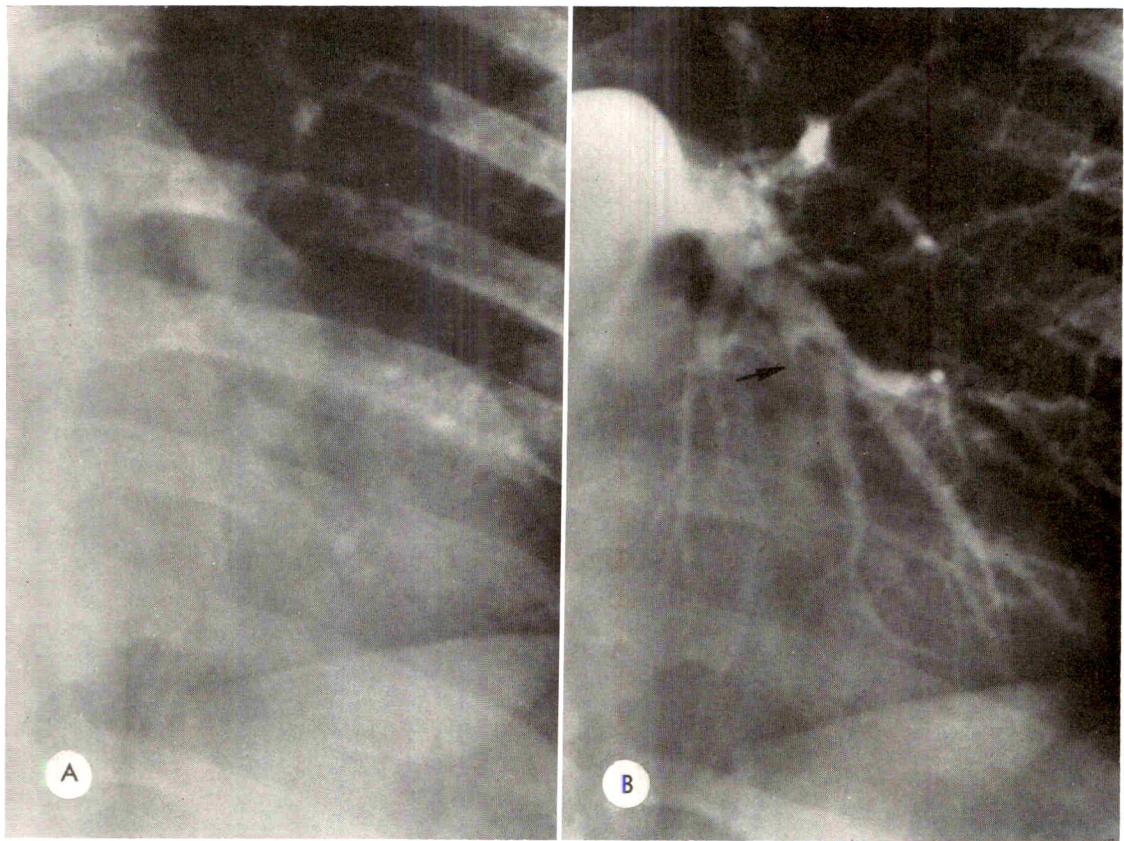


FIG. 8. Hypovascular lower lobe. (A) Overpenetrated roentgenogram prior to injection of contrast material. (B) Corresponding arteriogram.

the left lower lobe is obscured by the heart. As a result, conventional chest roentgenograms are not optimal for observing variations in vascularity. In retrospect, it was noted that the preliminary angiographic film provided a more unobstructed view of the lower lobes and demonstrated in several instances evidence of obstructed vessels (Fig. 7, A and B; and 8, A and B). On this basis, it is suggested that survey chest roentgenograms for pulmonary emboli should include an overpenetrated view of the lower lobes.

#### HEMODYNAMIC DATA

Right heart catheterization data were not as effective as the pulmonary angiogram in detecting emboli. Eleven of the patients who had normal resting pulmonary artery pressures had angiographic proof of their emboli. This was not surpris-

ing since greater than 60 per cent of the cross-sectional area of the pulmonary circulation must be occluded when large or middle sized arteries are involved before pulmonary hypertension is produced.<sup>13,15,23</sup> Nevertheless, determinations of the pulmonary artery pressure complement the angiogram, as was evidenced by the 3 patients who had pulmonary hypertension in the absence of angiographically demonstrated thromboemboli. This will occur especially in association with microemboli.

The response to unilateral pulmonary artery balloon occlusion was a more sensitive test than the resting pulmonary artery pressure. A normal patient responds to the occlusion by minimal change in pulmonary artery pressure and cardiac output and a marked decrease in vascular resistance. The rise in pulmonary artery pressure, following unilateral pulmonary artery balloon occlu-



sion is termed a positive response. This test should detect those instances of vascular obstruction where the cross-sectional involvement is not sufficient to elevate the main pulmonary artery pressure, yet extensive enough to prevent the diminution of vascular resistance that ordinarily accompanies diversion of the entire pulmonary flow into one lung. Two patients in this series had a normal resting pulmonary artery pressure, a normal angiogram, but a positive response to balloon occlusion. Four patients had a positive response and a positive angiogram.

An interesting phenomenon was the different responses noted following balloon occlusion in those patients with resting pulmonary hypertension. A mechanical obstruction was postulated for those patients who had further elevation of their pulmonary hypertension; and a humoral or

reflex basis when no further elevation resulted.<sup>5,9,10</sup> This hypothesis was further supported by the angiogram, which demonstrated relatively little anatomic involvement in those with a negative response. A positive response to unilateral pulmonary artery balloon occlusion in those patients with resting pulmonary hypertension is considered an indication for emergency inferior vena cava ligation.

#### THE ANGIOGRAM

The most striking finding on the pulmonary angiogram was the size and extent of central thrombus collection in the absence of corresponding clinical and plain film abnormality. When compared to conventional methods, it not only provided convincing proof of the presence of emboli, but enabled a better evaluation of the extent and location of the embolic process.<sup>22</sup> Angi-

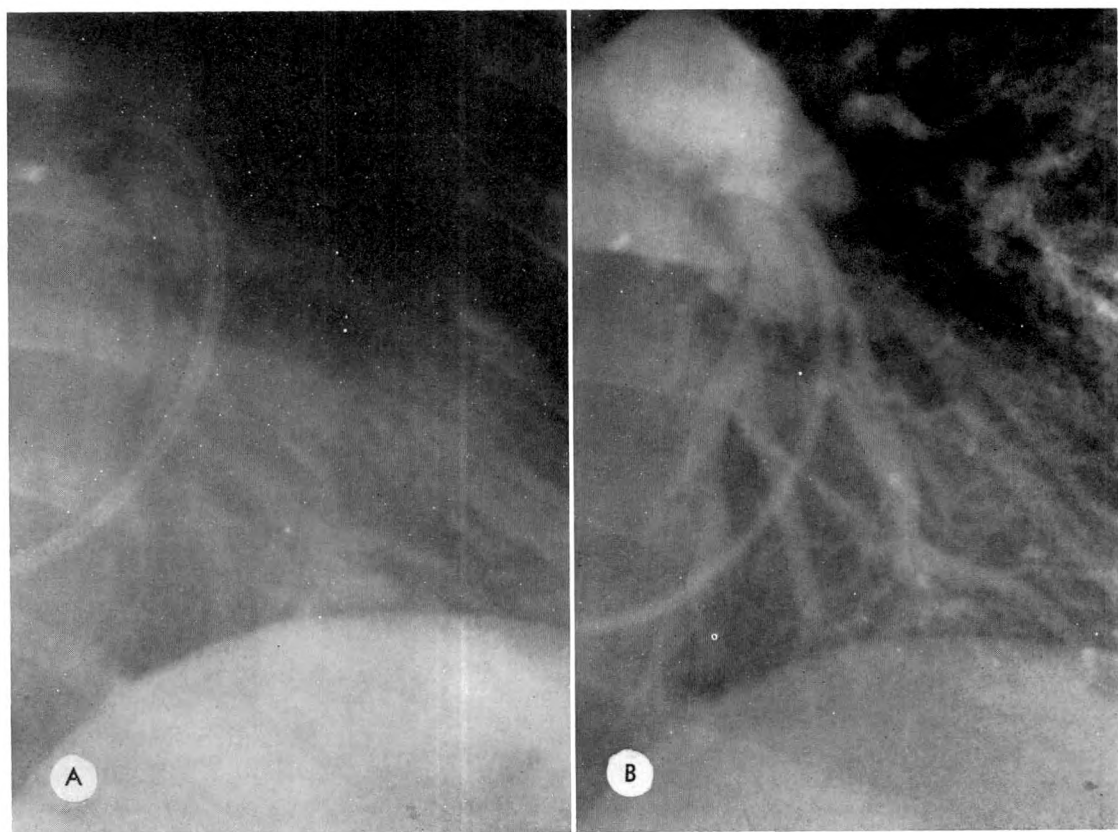


FIG. 9. Tortuous, distorted lower lobe vessels. (A) Overpenetrated roentgenogram prior to contrast material injection. (B) Corresponding arteriogram.

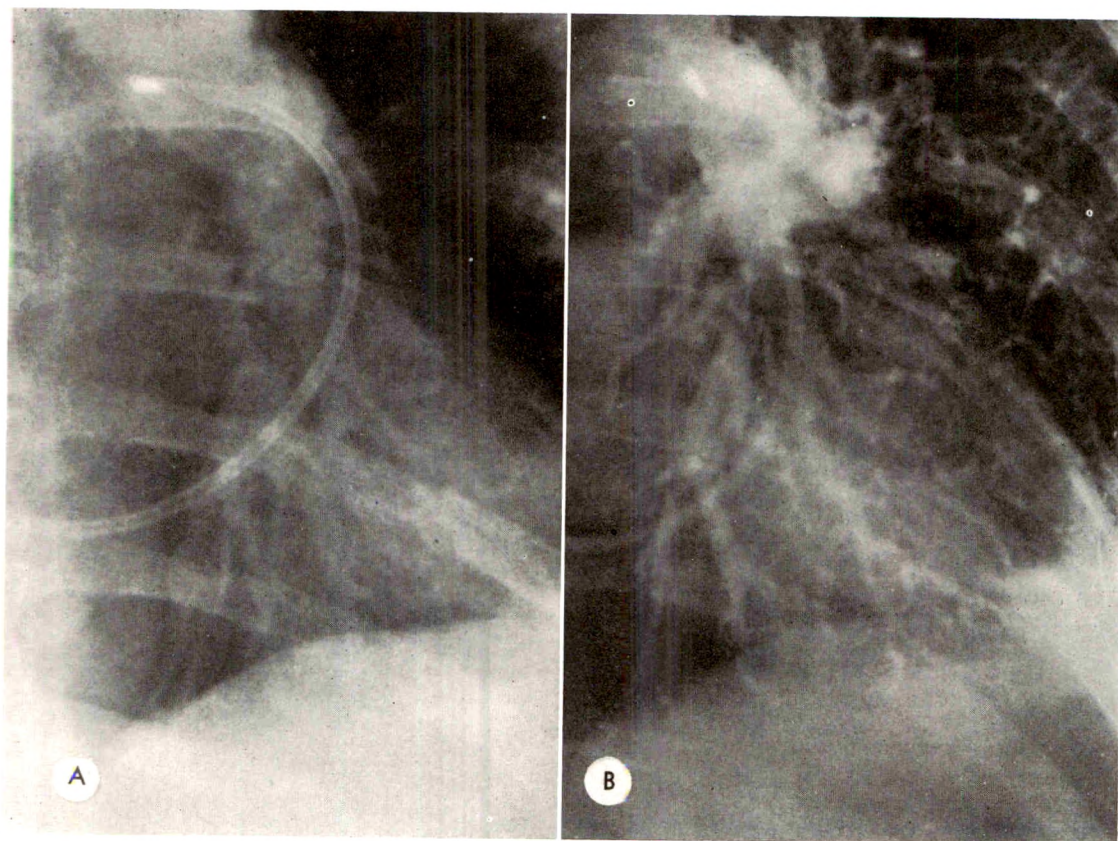


FIG. 10. Tortuous, distorted lower lobe vessels. (A) Overpenetrated roentgenogram prior to injection of contrast material. (B) Corresponding arteriogram.

ography is essential if pulmonary artery surgery is contemplated. Both the angiogram and the balloon occlusion study were helpful in identifying significant embolic disease in the contralateral lung in patients with unilateral chest pain.

The importance of optimal contrast definition on the angiogram cannot be overemphasized. Because of this, intravenous and right heart contrast injections were abandoned. Right atrial injections of contrast material preceded the pulmonary artery injection only in those cases where an intracardiac thrombus was suspected.

Angiographic diagnosis was more difficult when only small vessels were involved. The localized small vessel changes, described above, were very helpful in establishing a correct diagnosis and have been described previously.<sup>20</sup> Similar changes in the small vessels are seen in patients with

congenital heart disease who have high pulmonary vascular resistance, as demonstrated by wedge arteriography.<sup>1,4</sup> For pulmonary emboli the segmental distribution is a necessary requisite for diagnosis, since a similar but uniform and generalized vascular alteration may accompany severe heart failure with marked pulmonary hypertension, congenital heart disease with complicating increased precapillary resistance,<sup>14,20</sup> and reflex peripheral constriction of the pulmonary vessels. Similarly, a bilaterally symmetrical lower lobe involvement has been described in congestive failure and some cases of mitral stenosis.<sup>14,20</sup>

The shortened, plump, tortuous vessels supplying the area of embolization were frequently seen on the scout roentgenogram taken prior to the angiogram (Fig. 9, A and B; and 10, A and B). In several instances, it was possible to predict emboli by this ob-



servation. The alteration is not a simple one and can be mimicked by other parenchymal disturbances. However, as previously suggested, the overpenetrated roentgenogram of the lower lobes may be helpful in the plain film detection of pulmonary emboli.

#### SUMMARY

1. Pulmonary angiograms and data obtained from right heart catheterization, especially when combined with unilateral pulmonary artery balloon occlusion, were complementary and important diagnostic methods in the detection and definition of emboli in 38 out of 58 patients referred with a diagnosis of "possible pulmonary embolism."

2. The angiogram often demonstrates more extensive thrombus collection within either or both lung fields than would be suspected from conventional studies.

3. Large thrombi may be present without recognizable clinical or chest roentgenographic alteration. The embolic event may be followed by a silent period with clinical symptoms produced by later obstruction of more peripheral vessels. Similarly, there may be a lag in the embolic event and the production of a roentgen density. Serial roentgenograms are necessary to demonstrate transient parenchymal changes.

4. Emboli may be directly identified, either as arterial obstructions with abrupt cut-off of the affected vessel, a filling defect, or as localized arterial stasis. Additional diagnostic signs include segmental diminution in flow, poor capillary filling, avascularity, and diminished, or absent venous return from the affected area.

5. Multiple small emboli, which affect peripheral vessels, can be recognized by a segmental vascular change characterized by shortened, plump and wavy branch vessels, relatively sparse peripheral filling and tortuosity of the visualized distal branches.

6. Overpenetrated plain roentgenograms emphasizing the lower lobes in an attempt to recognize avascular areas or distorted

vessels are suggested in order to extend conventional diagnostic efforts.

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## ANGIOCARDIOGRAPHIC DIAGNOSIS OF PERICARDIAL EFFUSION\*

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**F**AILURE of pericardiocentesis to secure fluid does not necessarily mean that the diagnosis of pericardial effusion is in error. Herein is described the case of a 68 year old man who, after angiocardio-graphic studies over a 3 year period, was found to have dilated heart chambers and the classic findings of pericardial effusion—huge soft tissue densities surrounding the heart in frontal view.<sup>4-8</sup> Several pericardiocenteses failed to yield more than a few milliliters of fluid. Serial roentgenograms of the chest following the last pericardial tap, however, showed a left hydrothorax. Thoracentesis yielded fluid similar in character to that of the pericardium. The decrease in size of the cardiac silhouette was interpreted to be due to leakage of pericardial fluid into the pleural space caused by needle puncture of the pericardium.

The authors underscore the fact that when angiocardio-graphy demonstrates a pericardial effusion, this diagnosis should not be abandoned even when pericardiocentesis fails to yield fluid. Indeed, when there is associated venous hypertension, a prolonged circulation time, and normal-sized cardiac chambers, it is essential to differentiate cardiac tamponade from effusive-constrictive pericarditis.<sup>8</sup> In such a case, when pericardiocentesis does not really yield fluid or a pericardial cyst is suspected,<sup>3</sup> thoracotomy is warranted. Removal of pericardial fluid will alleviate cardiac tamponade if present,<sup>4,9,10</sup> and, at the same time, inspection and palpation of the pericardial layers will establish the diagnosis of effusive-constrictive pericarditis; pericardiectomy may then be curative.

### REPORT OF A CASE

A 68 year old white male (N.Y.H. No. 932623) was admitted on January 10, 1963, because of increasing shortness of breath. The patient had enjoyed excellent health until 1962, when chest pain and dyspnea began. He also had peripheral edema and hypertension, and was treated with diuretic and antihypertensive therapy, and then referred to this hospital.

Physical examination revealed a blood pressure of 175/100 mm. Hg. The pulse was irregular at a rate of 80 and respirations were 18 per minute. The neck veins were distended at 45 degrees, and fine rales were present at the right base. The left border of cardiac dullness was percussed at the anterior axillary line. A holosystolic (Grade 2-6) murmur was heard at the apex radiating to the axilla, and a harsh ejection (Grade 3-6) murmur was present at the base radiating into the neck. The pulmonic second sound was split physiologically. The liver was not enlarged and there was mild peripheral edema. Venous pressure was 170 mm. saline at the level of the right atrium. Circulation time (arm to tongue) was 45 seconds and the basal metabolism rate was plus 23. The electrocardiogram revealed atrial fibrillation with occasional ventricular premature contractions. Chest roentgenograms (Fig. 1, *A* and *B*) showed a huge cardiac silhouette. Intravenous angiocardio-graphs (Fig. 2, *A* and *B*), made on January 17, 1963, demonstrated enlargement of the cardiac chambers with massive pericardial effusion. The soft tissue density adjacent to the right border of the opacified right atrium measured 4.5 cm.; on the left it was 3.5 cm. beyond the left side of the opacified left ventricle. The left atrium measured 11 by 9 cm. (average normal is 8 by 5.5 cm.). Pericardiocenteses with electrocardio-graphic monitoring<sup>1,2</sup> were attempted, but no fluid was aspirated despite multiple punctures into the subxyphoid and

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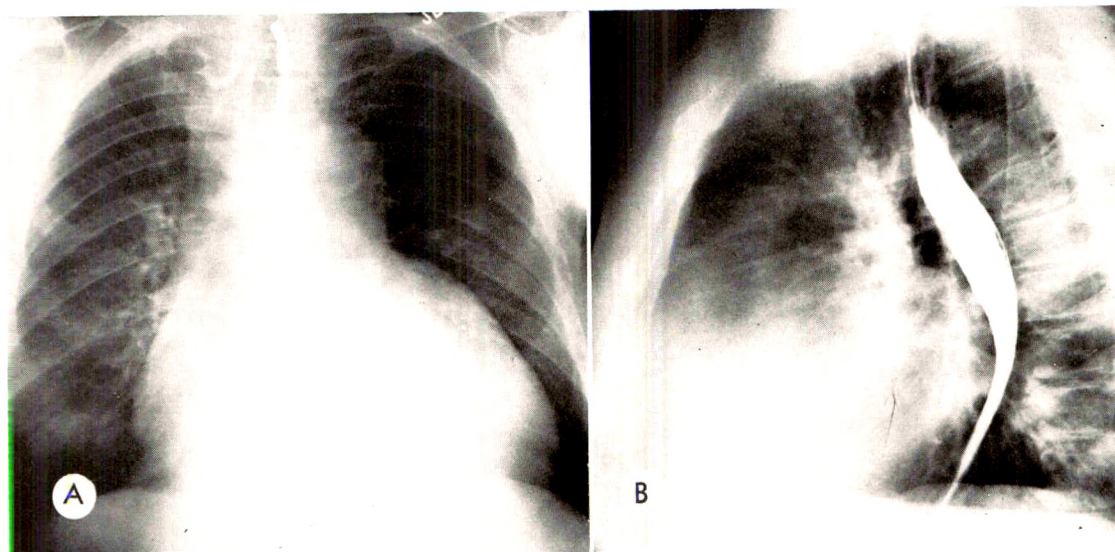


FIG. 1. (A) Frontal teleroentgenogram of the chest (January 14, 1963) showing a huge cardiac silhouette and dilated thoracic aorta. (B) Lateral esophagram (same day) showing displacement by an enlarged left atrium. Note obliteration of the retrosternal space.

peri-apical areas. Blood electrolytes, urinary catechol amines, and intravenous pyelography were unremarkable. He was discharged after digitalis and diuretic therapy, and disappearance of the venous hypertension; the cardiac silhouette was smaller than on admission (Fig. 3).

On February 14, 1966, the patient was readmitted because of increasing dyspnea and

fatigue. Examination showed enlargement of the heart extending to the left axilla (Fig. 4A). The pulse was irregular and the blood pressure was 145/95 mm. Hg. The systolic murmur at the apex and base of the heart was unchanged. An intravenous angiocardigram (Fig. 4B) on February 17, 1966, again demonstrated a huge pericardial effusion with dilatation of the cardiac chambers. The circulation time (dech-

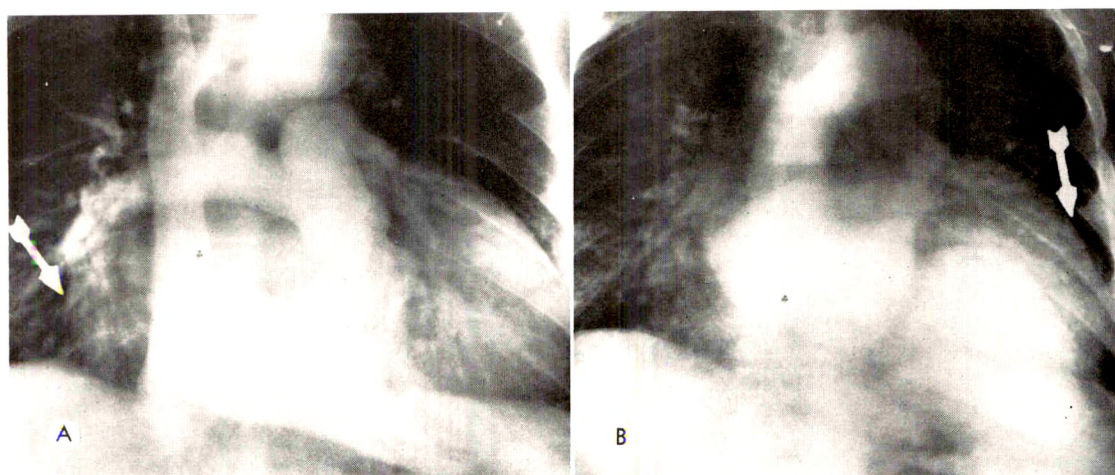


FIG. 2. (A) Frontal angiocardigram (January 17, 1963) showing pericardial fluid (arrow) beyond the opacified right border of the right atrium. Note the enlarged right ventricle, pulmonary artery, and branches. (B) Serial frontal angiocardigram showing enlargement of the left atrium and ventricle. Note the huge soft tissue density (arrow) beyond the left outer wall of the opacified left ventricle.



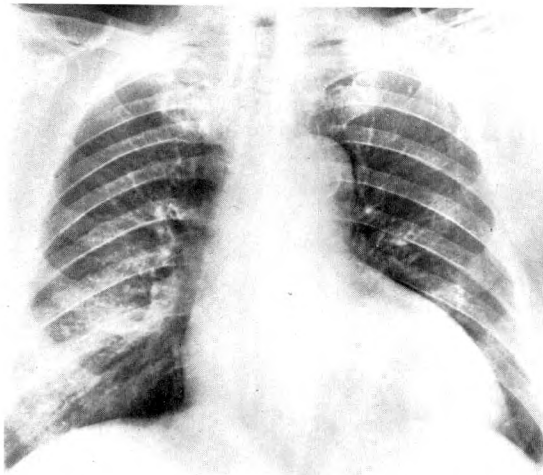


FIG. 3. Frontal teleroentgenogram of the chest (January 27, 1963) prior to discharge. Note that the cardiac silhouette became smaller following digitalis and diuretic therapy.

olin) was 32 seconds. The venous pressure was normal. Several attempts at pericardiocentesis yielded only 20 ml. of serosanguineous fluid obtained after puncture into the subxyphoid area. Smear of the fluid was negative for acid fast bacilli and fungi; and cultures were sterile. Papanicolaou studies of the pericardial fluid were also negative.

Two days later, several pericardiocenteses were again unsuccessful, but post-tap roentgen-

ograms (Fig. 5, *A* and *B*) showed a marked decrease in the size of the cardiac silhouette and a left hydrothorax. Thoracentesis of the left hemithorax yielded only 5 ml. of serosanguineous fluid similar in character to that obtained from pericardiocentesis. The remainder of the patient's course in the hospital was unremarkable; he improved and was discharged on February 26, 1966. The cardiac silhouette was decreased in size (Fig. 6).

He was seen regularly in the cardiac clinic until he was referred into the hospital on April 18, 1966, because of tachycardia, dyspnea, substernal pressure, abdominal swelling, and ankle edema. He improved after limiting salt ingestion and increasing the dosage of digitalis. The chest roentgenogram showed a huge cardiac silhouette of the same size as of the earlier admission (Fig. 4*A*). He was discharged on April 24, 1966.

#### DISCUSSION

It is natural to attribute enlargement of the cardiac silhouette (Fig. 1, *A* and *B*) to cardiac enlargement, especially when a patient has symptoms and signs of heart failure. Obliteration of the retrosternal space (Fig. 1*B*) in the absence of congenital or rheumatic heart disease etiology is, however, presumptive evidence of pericardial

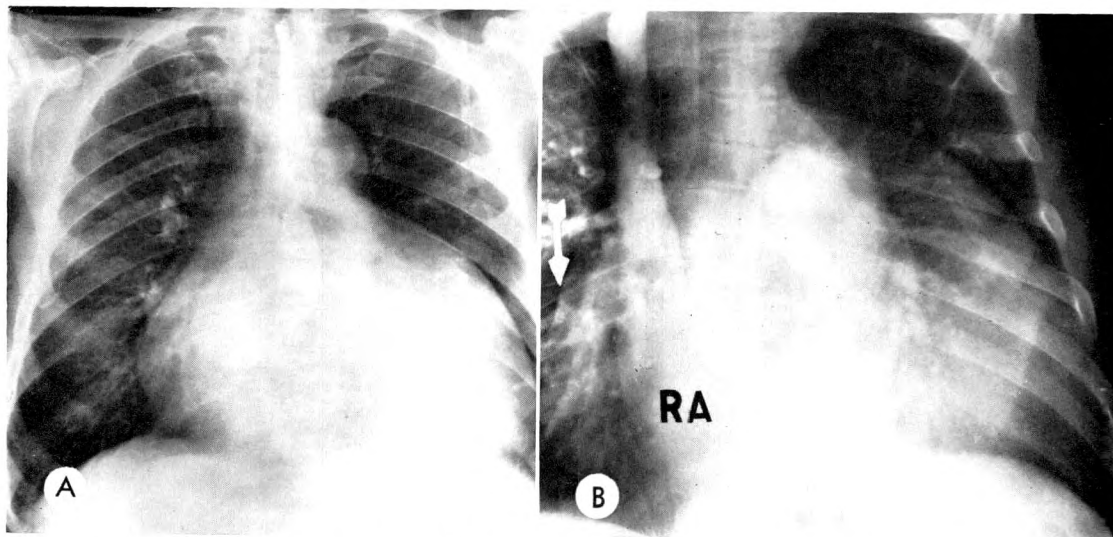


FIG. 4. (*A*) Frontal tele-esophagram again showing a huge cardiac silhouette upon re-admission on February 14, 1966. (*B*) Frontal angiogram (February 17, 1966) still shows a huge pericardial effusion and dilated cardiac chambers. Arrow points to outer margin of soft tissue density beyond the opacified right atrium (RA).

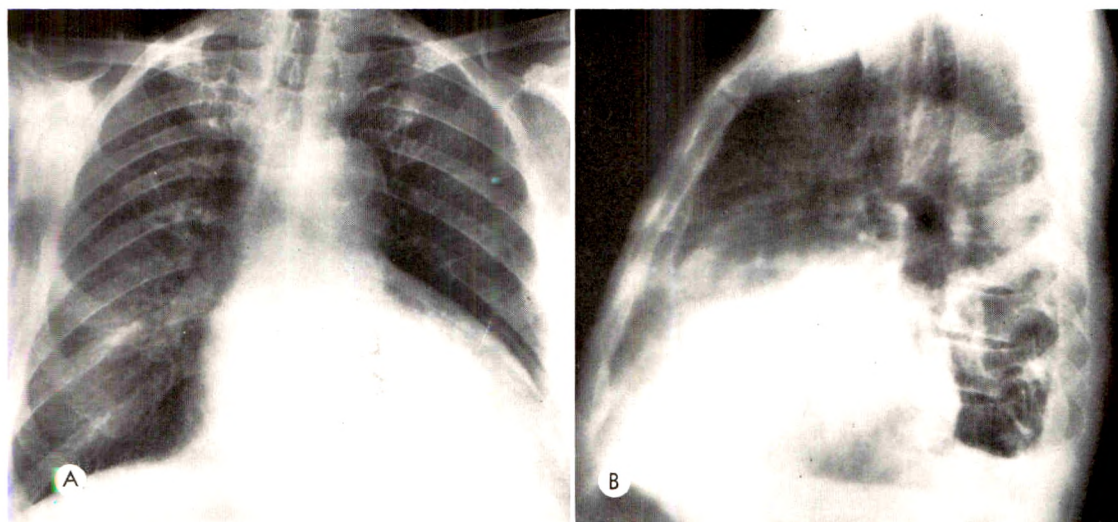


FIG. 5. (A) Frontal teleroentgenogram (February 24, 1966) showing the development of a left hydrothorax following pericardiocentesis. (B) Lateral roentgenogram also showing the pleural effusion.

effusion.<sup>5,7</sup> Angiocardiography, by visualizing the cardiac chambers, readily established the diagnosis of pericardial effusion (Fig. 2, A and B) and dilatation of the cardiac chambers due to heart failure.<sup>5,7</sup> Despite the inability to secure fluid after pericardiocentesis, the decrease in the size of the cardiac shadow (Fig. 3) following digitalis and diuretic therapy and the disappearance of venous hypertension substantiated the diagnosis of heart failure and pericardial effusion.

Chronic heart failure and even a larger cardiac silhouette were present 3 years later (Fig. 4A) and again angiocardiography revealed a pericardial effusion (Fig. 4B). Pericardiocentesis were unsuccessful, but fortunately serial roentgenography permitted detection of a left hydrothorax (Fig. 5, A and B; and 6). Although the etiology of the chronic heart failure could not be determined definitively in this case, the advanced age, a history of previous hypertension, the atrial fibrillation, and the dilated, tortuous, and elongated thoracic aorta are consistent with arteriosclerotic cardiovascular disease.

#### SUMMARY AND CONCLUSIONS

The case of a 68 year old man with symptoms and signs of congestive heart failure

and a huge cardiac silhouette, observed over a period of 3 years, is presented because, although angiocardiograms demonstrated an associated pericardial effusion, pericardiocentesis failed to substantiate it. Serial chest roentgenograms following the pericardiocentesis revealed the development of a left hydrothorax, and thoracentesis yielded a fluid similar to that of the pericardium. Following this, a decrease in the size of the cardiac silhouette permitted

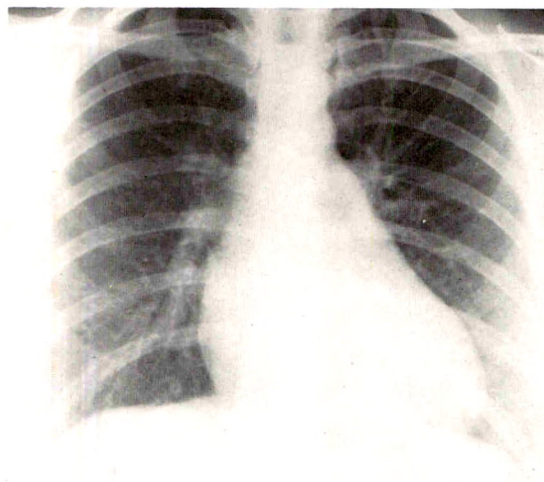


FIG. 6. Teleroentgenogram of the chest on discharge from the hospital (February 26, 1966) showing the decreased size of the heart following escape of pericardial fluid into the left pleural space.



the assumption that the pericardiocenteses established a pericardial left pleural window and allowed drainage of the pericardial effusion. Thus, the reliability of angiocardiology for the diagnosis of pericardial effusion was reaffirmed. The case also emphasizes the importance of serial roentgenography of the chest for a diagnosis of the creation of a pericardial pleural window.

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## CATHETER ARTERIOGRAPHY IN THE EVALUATION OF ABDOMINAL AORTIC ANEURYSMS\*

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THE investigation of abdominal aortic aneurysms by angiography has raised two controversial issues—one pertaining to the need for such study, and the other to the most suitable angiographic technique. We have found arteriography valuable in evaluating patients for therapy, planning surgery, and weighing the prognosis of treatment of abdominal aortic aneurysms. A technique based primarily on retrograde femoral catheterization has been found to be safe and relatively simple, and regularly to produce the high contrast, detailed arteriograms which are necessary to provide useful information.

### MATERIALS AND METHODS

In the past 2 years we have studied 52 patients with abdominal aortic aneurysms. In addition, angiograms were obtained on 5 patients and failed to confirm a clinical impression of aneurysm. Many patients with diffuse aortic dilatation, small localized bulges in an atherosclerotic aorta, or dissecting aneurysm were also examined, but are not included in this report.

When both femoral pulses were unimpaired, the right common femoral artery was chosen as a puncture site because of the usually straighter course to the aorta.<sup>9</sup> After local anesthesia with 2 per cent lidocaine, a 16 gauge thin-walled Seldinger needle was inserted percutaneously just above the inguinal crease. Under fluoroscopic monitoring, a guide wire, .045 inch in diameter with a 3 cm. flexible tip, was passed through the needle into the common iliac artery if no resistance was encountered. If this guide wire did not advance absolutely freely, it was replaced by another with a long flexible tip supported by

a cable and curved at the end.<sup>1</sup> No effort was made to advance the guide wire into the aorta. The needle was then replaced by a polyethylene (PE 240) catheter, tapered to the guide wire with a J-tip,<sup>2</sup> about 1 cm. in diameter and three pairs of side holes proximal to the J. When the tip had been advanced beyond the end of the guide wire and the J had reformed in the iliac artery, the catheter was introduced into the aorta and advanced through the aneurysm.

A complete study included injections above, into and below the aneurysm, with serial roentgenograms demonstrating the aorta and its branches from the level of the diaphragm to the lower leg. Renografin 60 was used as the contrast medium, in doses of 25–50 cc., administered at a rate of 15–30 cc./sec. by an automatic injector. The total dose of contrast agent ranged from 100 to 250 cc. per study. Vasodilator drugs or tourniquet ischemia<sup>10</sup> were employed as needed to increase the flow of blood. Arch aortography, usually via the right axillary artery, was performed as a separate procedure when indicated.

In the infrequent cases in which a needle, guide wire or catheter could not be introduced easily via the femoral route, the alternate approach was high translumbar puncture with a catheter needle. This was usually combined with noncatheter femoral arteriography and countercurrent injection to visualize the distal aorta and iliac arteries.

### RESULTS

In 50 of the 57 cases, a catheter could be introduced successfully on the side selected initially. In 4 cases the opposite side had to be used. In 3 cases, neither iliac artery

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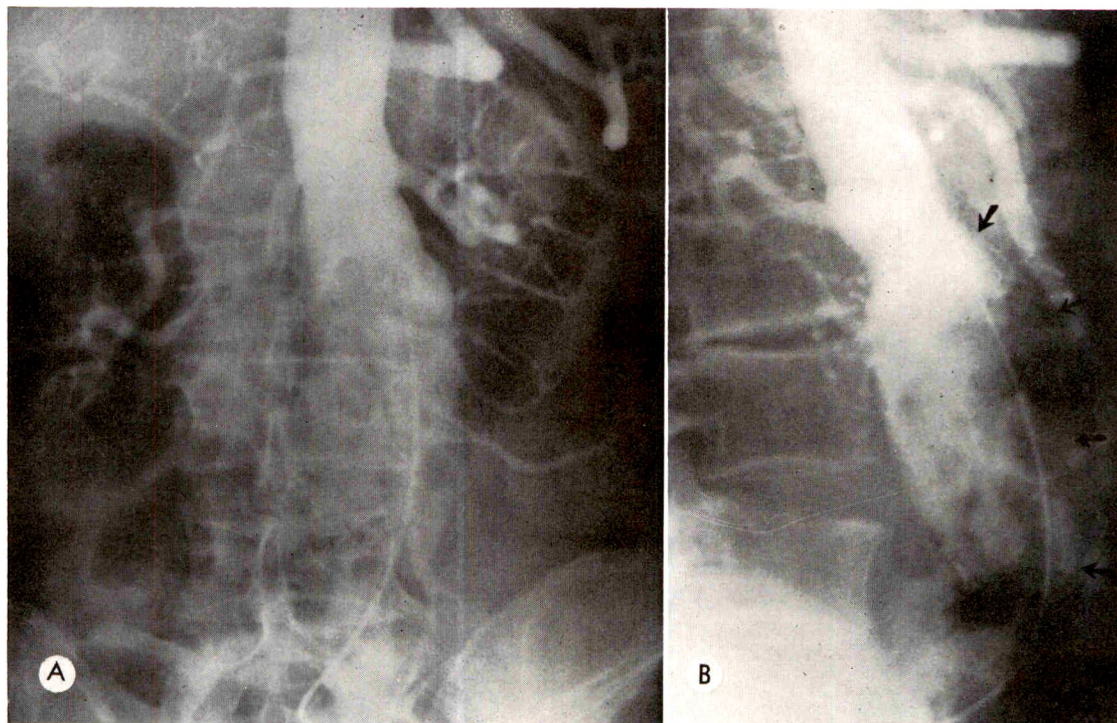


FIG. 1. Fusiform aortic aneurysm demonstrated by retrograde femoral catheterization and biplane angiography. Layering of contrast material prevents full appreciation of the size of the aneurysm in the frontal view (*A*), but in the lateral view (*B*), the anterior margin is outlined both by calcium (arrows) and the overlying superior mesenteric artery.

could be negotiated, and after bilateral femoral arteriography and countercurrent aortography, translumbar aortography was performed. No clinical complications were encountered, although 2 instances of extraluminal passage of the catheter were demonstrated roentgenographically. Satisfactory studies were obtained in all cases.

The following information was then available for further evaluation of the patient:

1. *Size.* The diameter of the aneurysm was computed by analyzing the associated soft tissue mass and calcification on plain roentgenograms, as well as the contrast column in the aorta and superior mesenteric artery. Because of the layering of contrast material posteriorly (Fig. 1, *A* and *B*) and the presence of intraluminal thrombus, the width of opacified aorta in the frontal view was not considered to represent the width of the aneurysm. However, even when no

calcium was visible in either wall, the size of the aneurysm could be estimated accurately in the lateral or oblique view by noting the position of the superior and inferior mesenteric arteries.

2. *Location.* While most aneurysms were located between the renal arteries and the aortic bifurcation, we encountered instances of diffuse aortic dilatation and of aneurysms on both sides of the renal artery. Sometimes, the higher aneurysm was the more significant (Fig. 2, *A*, *B* and *C*).

3. *Renal arteries.* The distance between the lowest renal artery and the aneurysm was measured to estimate the space available for cross clamping the aorta at surgery. This distance was sometimes foreshortened in the frontal view, and biplane views provided greater accuracy. The greatest problem was provided by double renal arteries, the lower of which frequently arose very close to, or even from the wall of the aneu-



rysm (Fig. 3, *A*, *B* and *C*). In addition, stenotic lesions (Fig. 4) or aneurysms (Fig. 7; *A* and *B*) of the renal arteries were found sometimes.

4. *Inferior mesenteric artery*. Occlusion of the inferior mesenteric artery was shown in a number of cases by retrograde filling of the left colic artery from the superior mesenteric artery (Fig. 5, *A* and *B*). Non-visualization of the inferior mesenteric

artery did not, by itself, prove occlusion, since the vessel often arose from the most anterior portion of the aneurysm while the contrast material layered out posteriorly.

5. *Iliac arteries*. While some aneurysms ended abruptly at the aortic bifurcation, others were associated with dilated iliac arteries. It was possible to estimate the condition of the vessels at the site of distal anastomosis of the graft. The patency of

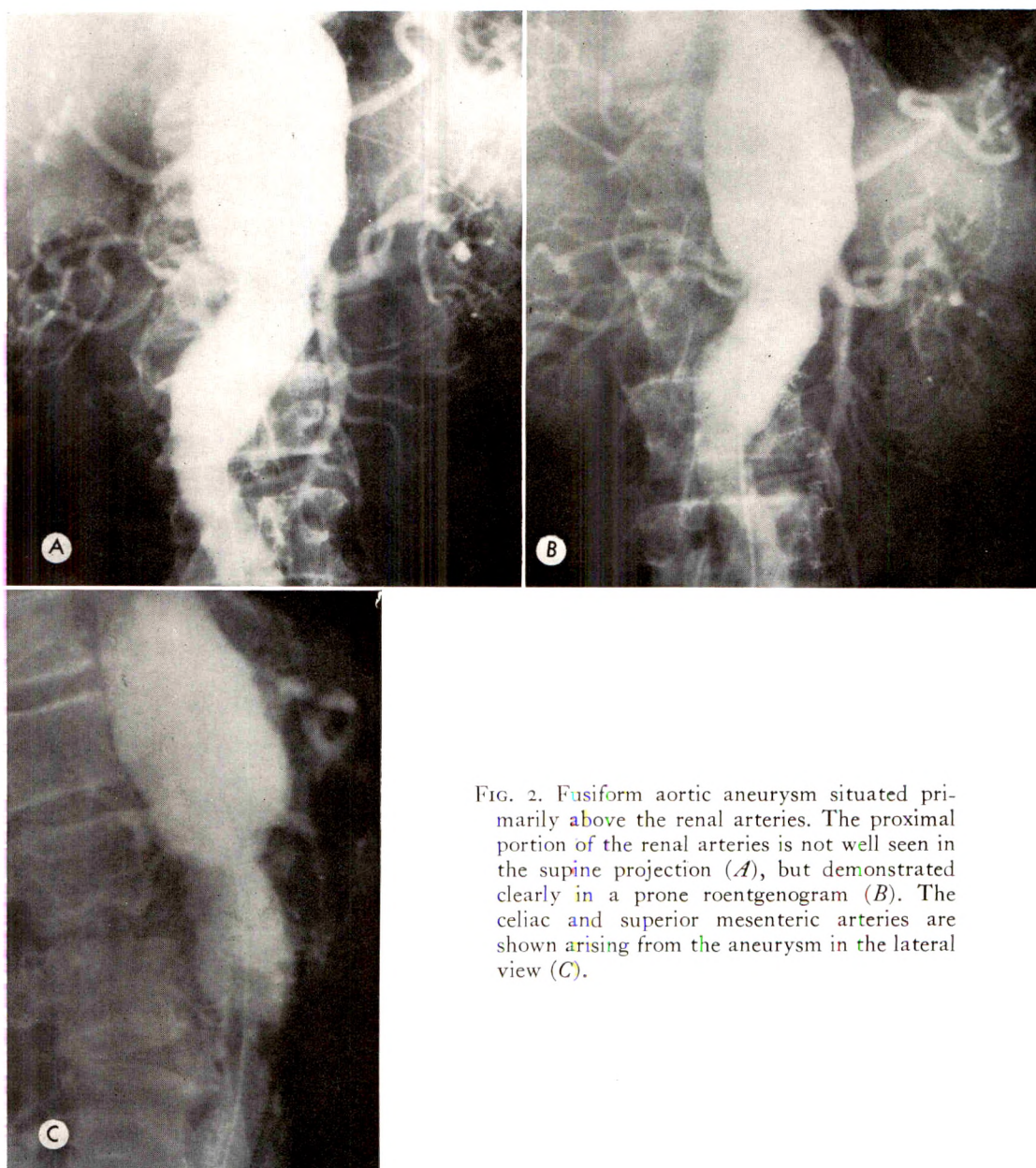


FIG. 2. Fusiform aortic aneurysm situated primarily above the renal arteries. The proximal portion of the renal arteries is not well seen in the supine projection (*A*), but demonstrated clearly in a prone roentgenogram (*B*). The celiac and superior mesenteric arteries are shown arising from the aneurysm in the lateral view (*C*).



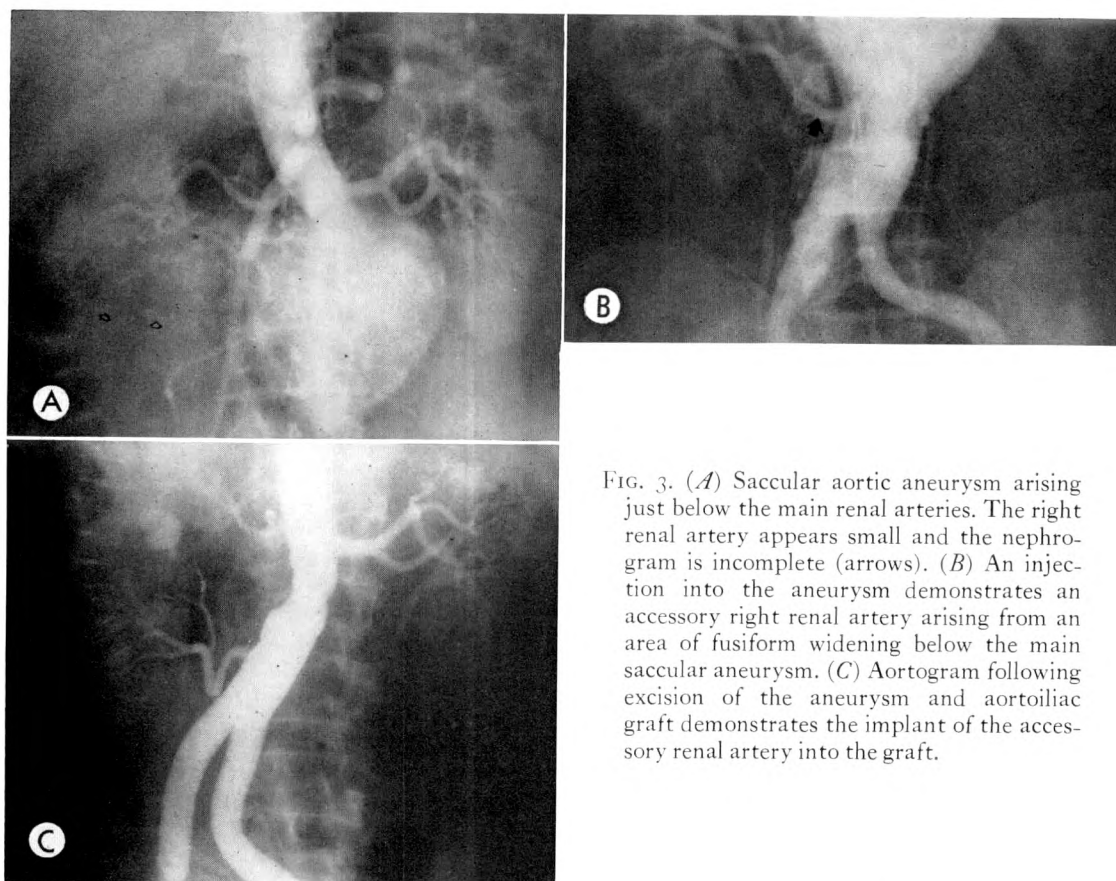


FIG. 3. (A) Saccular aortic aneurysm arising just below the main renal arteries. The right renal artery appears small and the nephrogram is incomplete (arrows). (B) An injection into the aneurysm demonstrates an accessory right renal artery arising from an area of fusiform widening below the main saccular aneurysm. (C) Aortogram following excision of the aneurysm and aortoiliac graft demonstrates the implant of the accessory renal artery into the graft.

the internal iliac arteries could be evaluated, as well as whether they could be preserved.

6. *Arteriovenous communications.* A patient with massive rupture of an aortic aneurysm into the vena cava was too ill to permit angiography. However, in another patient with a large aneurysm, it was possible to demonstrate a smaller arteriovenous communication into the ascending lumbar vein (Fig. 6, A and B).

7. *Associated arterial disease of the lower extremities.* The presence of separate aneurysms or of significant occlusive disease of the femoral arteries was demonstrated frequently (Fig. 7, A and B). Satisfactory visualization of lesions of the popliteal arteries (Fig. 8), and more distally, usually depended upon the employment of vasodilatation by drugs or reactive hyperemia.

8. *Associated disease in the thoracic aorta*

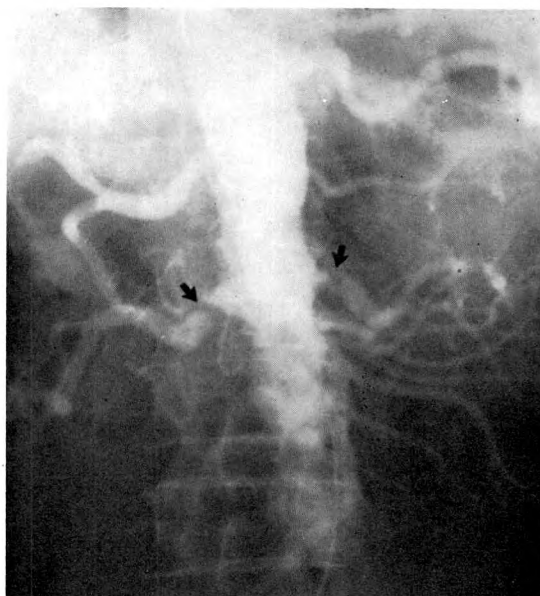


FIG. 4. Bilateral renal artery stenosis (arrows) in patient with fusiform aortic aneurysm. The 8 cm. wide aneurysm is incompletely filled in this view because of contrast layering.

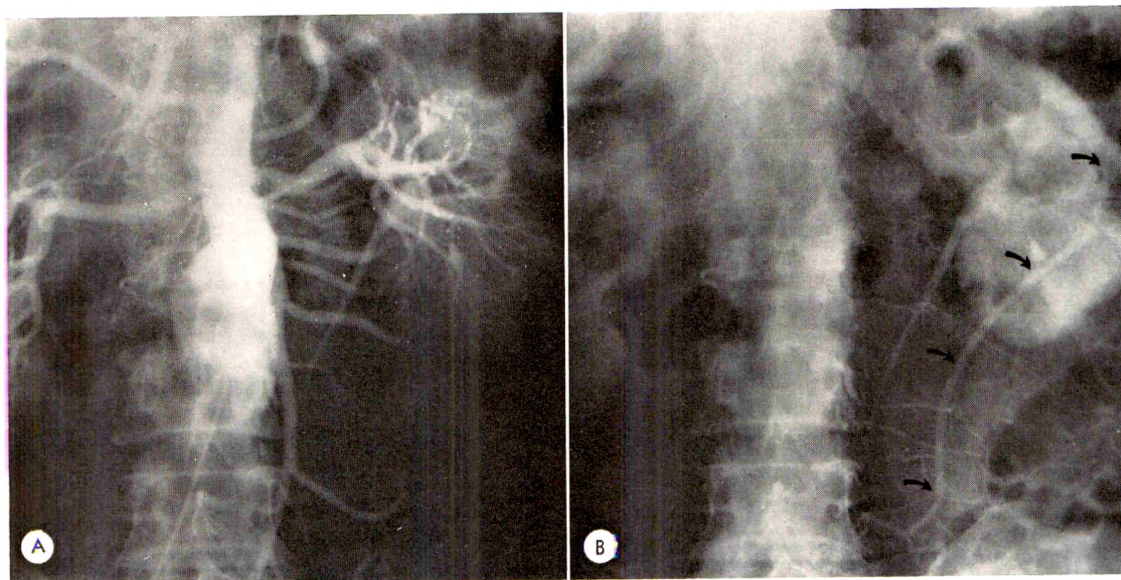


FIG. 5. (A) Sacular infrarenal aortic aneurysm. (B) Occlusion of the inferior mesenteric artery is shown by the retrograde filling of this vessel from the superior mesenteric artery via the left colic branch (arrows).

and brachiocephalic vessels. Lesions producing significant impairment of cerebral circulation, and thus increasing the risk of major abdominal surgery, were sometimes present. While these might have been

demonstrated by advancing the femoral catheter into the thoracic aorta for another injection, it was usually elected to obtain a higher quality brachiocephalic arteriogram on another day by inserting a shorter

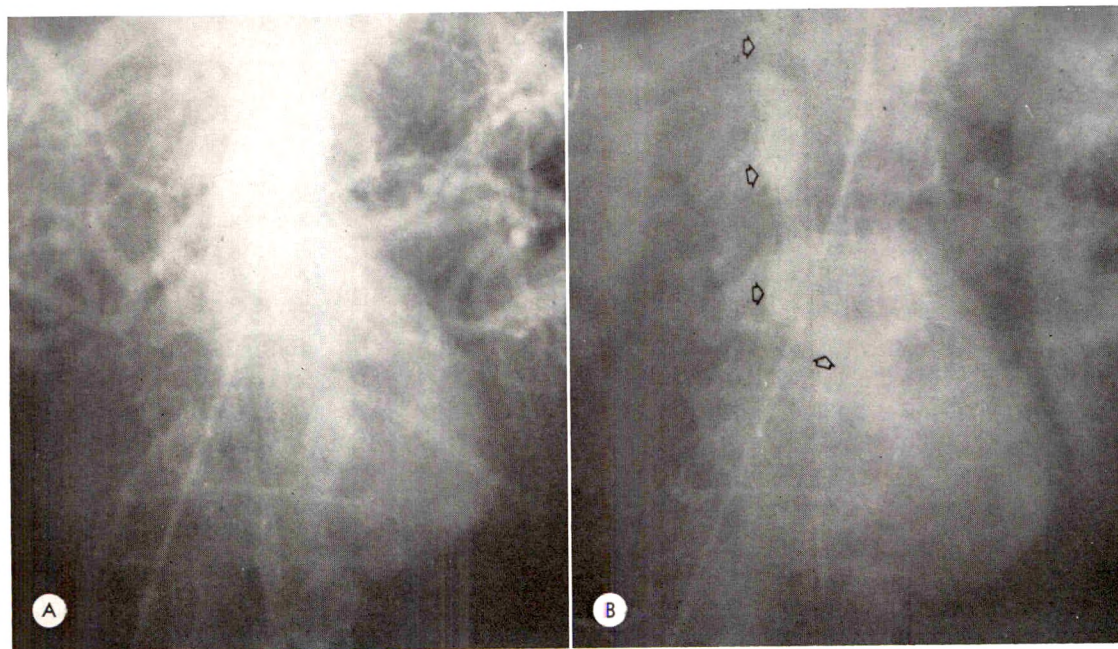


FIG. 6. (A) Large sacular aortic aneurysm. (B) Later roentgenogram in the same series demonstrates filling of a dilated, ascending lumbar vein (arrows) while the aneurysm is still partly opacified. This finding strongly suggests the presence of an arteriovenous communication.



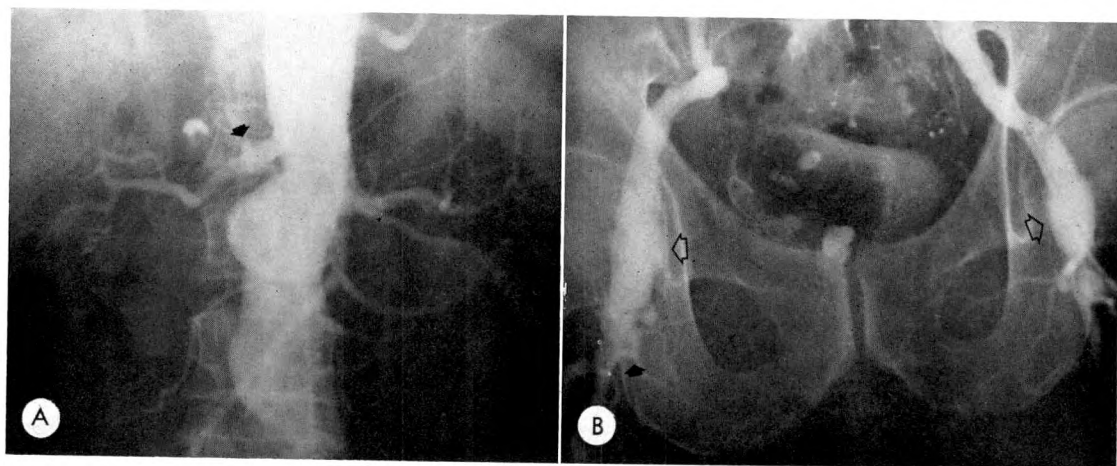


FIG. 7. Saccular and fusiform aortic aneurysm as well as multiple other arteriosclerotic changes. (A) Right renal artery aneurysm (arrow). (B) Fusiform dilatation of both common femoral arteries (white arrows) and occlusion of both superficial femoral arteries (black arrows).

catheter into the ascending aorta from the right axillary artery (Fig. 9, A and B).

#### DISCUSSION

The controversies regarding the indications and techniques for aortography in abdominal aortic aneurysms cannot be discussed in an isolated manner. Unless a

method is employed which is not only safe but informative, the routine use of aortography can hardly be justified. In most instances the diagnosis of aortic aneurysm is clinically quite definite, and a procedure which merely demonstrates the aneurysm without providing additional anatomic data is unnecessary. For this reason, we have ruled out the use of remote injections, whether intravenous,<sup>4,19,20</sup> right atrial,<sup>5</sup> or countercurrent left brachial,<sup>11,13</sup> as they provide inadequate detail.

Catheterization of the abdominal aorta via the brachial<sup>18</sup> or axillary<sup>8,15</sup> route is often quite difficult in the presence of a tortuous vessel. The justification of this circuitous approach would require proof, rather than glib assumption, that femoral catheterization is more dangerous. The translumbar approach has a long record of safety,<sup>3,14</sup> but unfortunately allows little flexibility in the selection of an injection site. Furthermore, there is a slight risk that fibrosis secondary to a clinically nonapparent hematoma at the aortic puncture site may complicate subsequent surgery. Countercurrent femoral noncatheter aortography usually fails to demonstrate the vessels proximal to the aneurysm.

Retrograde femoral catheterization must be performed with great care in order to avoid a high incidence of dissection of



FIG. 8. Popliteal aneurysm in patient with fusiform aortoiliac aneurysm. Because of the extremely slow circulation time, this was demonstrable only by angiography after tolazoline infusion.

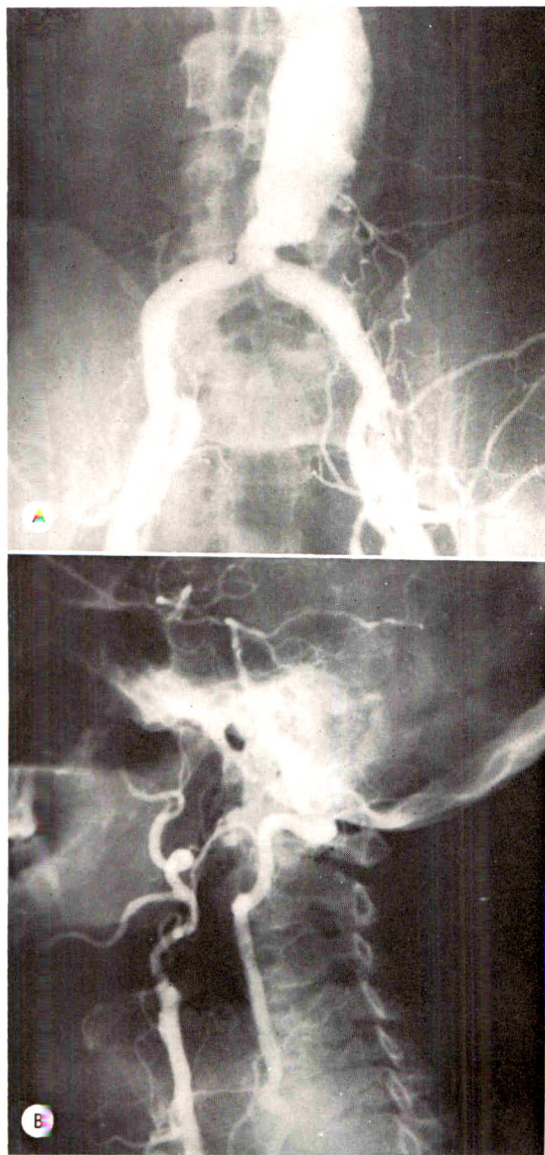


FIG. 9. Association of aortic aneurysm with cerebrovascular disease. (A) Distal aortic aneurysm with stenosis at the aortic bifurcation. (B) Innominate arteriogram with injection via the right axillary artery shows complete occlusion of the origin of the right internal carotid artery (arrow).

arteriosclerotic vessels by the guide wire or catheter.<sup>7</sup> While most perforations of the intima are undoubtedly asymptomatic, the possibility of catastrophe always lurks in the background. However, with the meticulous avoidance of force and the use of curved flexible guide wires and polyethy-

lene J-catheters, the risk of complications appears to be quite minimal. Straight catheters and catheters made of firm materials such as teflon or nylon have no place in this technique. Furthermore, the method is not recommended for the physician who is just beginning to become familiar with catheter manipulation, or who does not have a good image intensification unit in the room in which the study is performed. Under these circumstances, a combination of translumbar aortography and noncatheter femoral arteriography would be much safer. However, with the application of modern skills and equipment for retrograde arterial catheterization, the dangers which have been stressed so often in the past have diminished greatly.

While there is general agreement that doubtful clinical diagnosis of aortic aneurysm is an indication for angiography, many authors feel that there is no need for arteriography in the definitively diagnosed case.<sup>3,6,12,16</sup> Some feel that its use should be limited to asymptomatic aneurysms,<sup>21</sup> particularly if they are small.<sup>17</sup>

We believe that angiography by a method which proves good anatomic detail is desirable in all patients who are

TABLE I  
SIGNIFICANT ANGIOGRAPHIC FINDINGS IN 52 PATIENTS  
WITH ABDOMINAL AORTIC ANEURYSMS

Extension above main renal arteries	2
Extension to within 1.5 cm. of main renal artery	11
Accessory renal artery arising from aneurysm	2
Separate renal artery aneurysm	1
Occlusion or marked stenosis of renal artery	9
Occlusion of inferior mesenteric artery	12
Arteriovenous fistula from aneurysm	1
Extension into common iliac artery	14
Separate iliac, hypogastric, femoral or popliteal artery aneurysm	7
Occlusion or marked stenosis of common or external iliac artery	7
Occlusion of common or superficial femoral artery	11
Occlusion of popliteal artery	9
Occlusion or marked stenosis of carotid or vertebral artery	6
Separate aneurysm of thoracic aorta	3



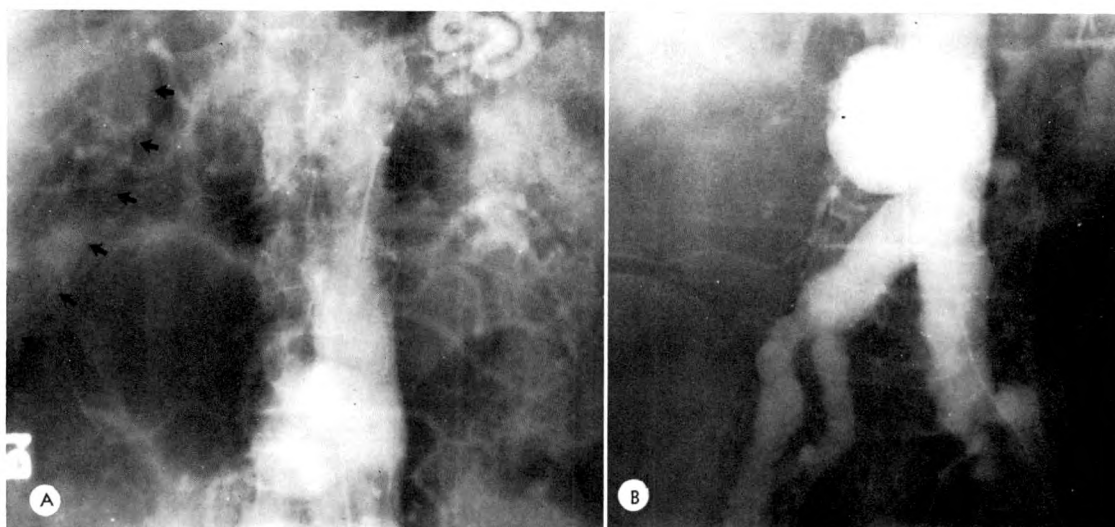


FIG. 10. Leaking aortic aneurysm. (A) Marked displacement of the right kidney (arrows) is associated with a saccular aneurysm bulging to the right. (B) From the blister-like appearance of this aneurysm, one may infer that its wall is likely to be quite thin (courtesy of Dr. Lester V. Nickrosz, Boston Veterans Hospital).

being prepared or considered for elective or semi-elective aneurysm repair. In the relatively good risk patient, there will be valuable information to help the surgeon plan his operation as effectively as possible. In the patient who is a doubtful surgical candidate, a more complete knowledge of the exact nature of the lesion and of associated vascular disease permits a more intelligent choice of operative or medical management (Table 1). Even when a leaking aneurysm is present, arteriography can be performed safely (Fig. 10, A and B). Only in the dire emergency presented by an acutely ruptured aneurysm should aortography be bypassed.

#### SUMMARY

Retrograde femoral artery catheterization for abdominal aortic aneurysm, when performed by a careful technique, with curved guide wires and polyethylene J- catheters, was successful in 54 of 57 patients. No clinical complications were encountered. The use of this method gave information not only as to the size and location of the aneurysm, but also the presence of aberrant renal artery stenosis or aneurysm, the condition of the inferior

mesenteric artery, the distal extent of the aneurysm, the presence of arteriovenous communication, and the existence of significant occlusive disease in the lower extremities. Arch aortography for evaluation of the vessels to the head was also performed frequently. Unlike the low-detail techniques such as intravenous aortography, which are mainly useful in confirming the presence of an aneurysm in the rare case where there is clinical doubt, the retrograde femoral technique safely provides sufficient anatomic information to justify its use in the evaluation of the patient being considered for aneurysm repair.

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## CAVOGRAPHY FOLLOWING PLICATION OF THE INFERIOR VENA CAVA\*

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IN RECENT years, there has been increasing interest in the prevention of pulmonary embolization by surgical procedures designed to compartmentalize the vena cava.<sup>3,5,9,10</sup> It has been hoped that such therapy might prevent massive and fatal embolism, yet avoid the postoperative sequelae which have been ascribed to ligation.<sup>2,4,6,7,8</sup>

The surgical procedures currently employed to this end fall into 3 general groups: plication with mattress sutures, with metal staples, or by external clipping (Fig. 1). All are designed to reduce the lumen of the inferior vena cava to multiple small channels which will entrap circulatory emboli larger than 2–3 mm. in size.

In view of the increasing numbers of patients undergoing such surgical procedures, it is important that the radiologist be able to evaluate the functional status of the vena cava following plication; with population mobility as it is, radiologists may be called upon to evaluate the postoperative status of patients who have been operated upon elsewhere. A general description of the post-plication appearance of the inferior vena cava has not been provided in the roentgenologic literature.

### MATERIAL AND METHODS

Fifty-five patients were treated at The Johns Hopkins Hospital with plication of the inferior vena cava in the past 5 years. This communication concerns 22 of those patients in whom 28 inferior vena cavographies were performed. Their average age was 52 years, ranging from 21 to 76 years. There were 12 white and 10 Negro patients with equal numbers of males and females.

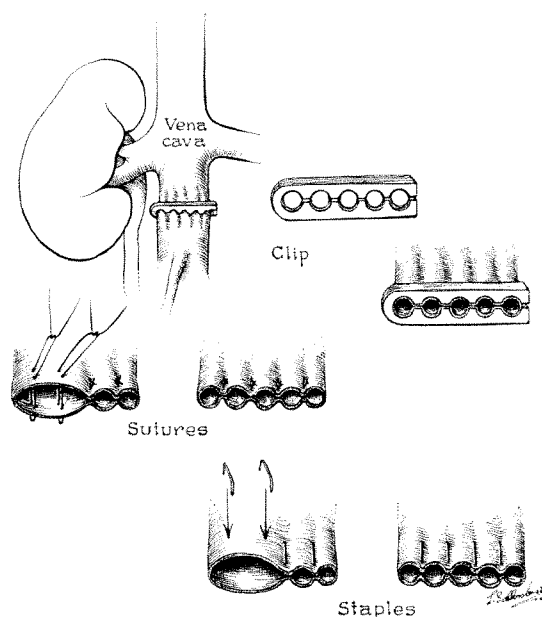


FIG. 1. Diagrammatic demonstration of types of plication procedures.

The diagnosis of pulmonary embolization, usually recurrent, was well established preoperatively in every case. Most of these patients had a preoperative history of phlebitis or varicosities. Three had sustained recent trauma or crush injury to the legs. Many had some form of heart disease. Selection of patients for postoperative cavography was arbitrary. Some were chosen because of clinical indications that the plication was obstructed—in others the procedure was done in order to confirm caval patency and to visualize the functioning plication site.

The technique of inferior vena cavography consisted of unilateral or bilateral femoral venapuncture, usually with the 18

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gauge Seldinger or the 17 gauge polyethylene sheathed needle. Hand injection of 60 cc. of contrast material with serial biplane film studies over a 10 second period was the usual procedure employed. Less contrast material was used if there was edema of the legs or if free flow of blood could not be obtained following venapuncture.

#### ROENTGENOGRAPHIC FINDINGS

The roentgenographic findings can be divided into 4 basic types: (1) normal functioning plication; (2) partial obstruction at the plication site; (3) complete thrombosis at the plication site; and (4) complete or partial breakdown of the plication site. Representative examples are illustrated below.

*Normal.* This patient was a 54 year old white female with a history of rheumatic heart disease with mitral stenosis, atrial fibrillation, and multiple pulmonary emboli. The vena cavograms shown in Figure 2, A and B were made 3 weeks after placement of a teflon clip on the vena cava. Relatively free flow of contrast material through the channels formed by the clip was obtained. There was no significant collateral circulation. Postoperatively, the pa-

tient continued to have symptoms and signs of recurrent emboli, probably from sources above the plication site.

*Partial occlusion* of the plication site was seen in 2 cases. The vena cavograms of one of them are shown in Figure 3, A and B. The patient was a 51 year old white female with multiple pulmonary emboli but without signs or symptoms of phlebitis. A persistent defect along the right side of the vena cava, which probably represented trapped thrombus not producing significant obstruction, was visualized. Two years after the plication procedure and vena cavography, the patient developed pulmonary infarction, again without clinical evidence of phlebitis.

*Complete thrombosis* presented a variable roentgenographic appearance according to the major collateral branches utilized in bypassing the obstructed segment. Angiographic visualization ("reconstitution") of the vena cava above the plication site is not unusual. Some of the varieties of collateral flow in complete caval obstruction are shown in Figures 4, A-D; and 5, A and B.<sup>1</sup>

*Breakdown* of the plication due to the sutures pulling out has been previously de-

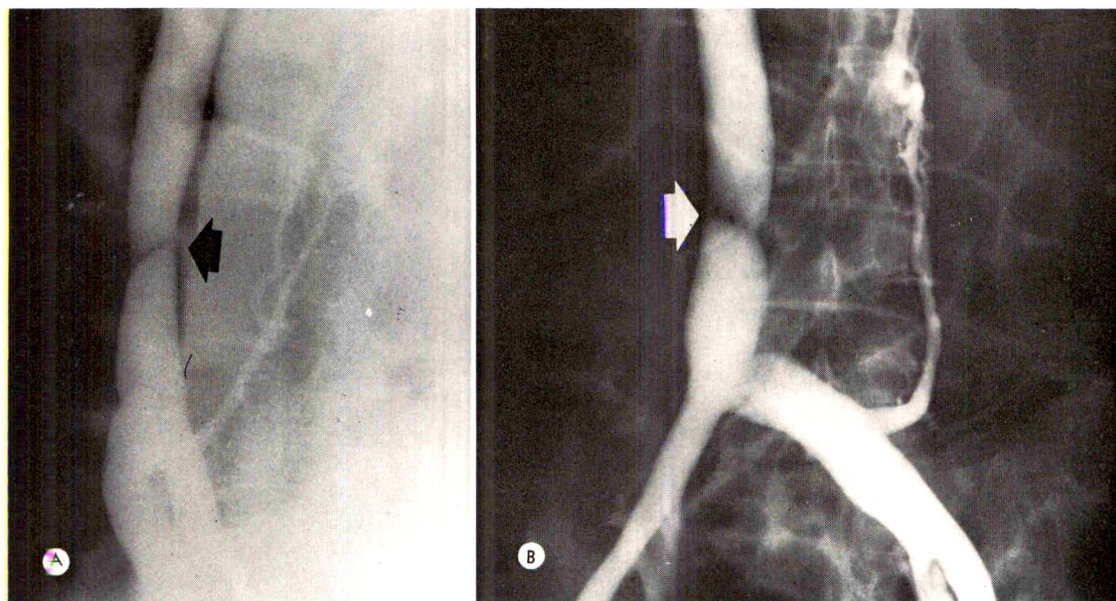


FIG. 2. (A and B) Functioning vena cava following placement of external teflon clip. The individual channels of the patent plication can be clearly identified in the anteroposterior projection (arrows).



scribed.<sup>1</sup> Figure 6, *A* and *B* shows vena cavograms of 1 of the 2 patients in whom breakdown of the plication site evidently occurred. Both of these patients had evidence of recurrent pulmonary embolization.

In some instances postoperative signs and symptoms of venous insufficiency in the lower extremities correlated poorly with roentgenologic findings of patency or thrombosis of the vena cava. Two patients who clinically were thought to have func-

tioning plications because of their benign postoperative courses were subsequently shown to have complete obstruction and rather poor collateralization (Fig. 7, *A* and *B*).

Conversely, the patient shown in Figure 8, *A* and *B* had no signs or symptoms of venous insufficiency in the lower extremities prior to an ankle fracture complicated by pulmonary embolus. The vena cavograms following plication showed narrowing at the plication site but no thrombus,

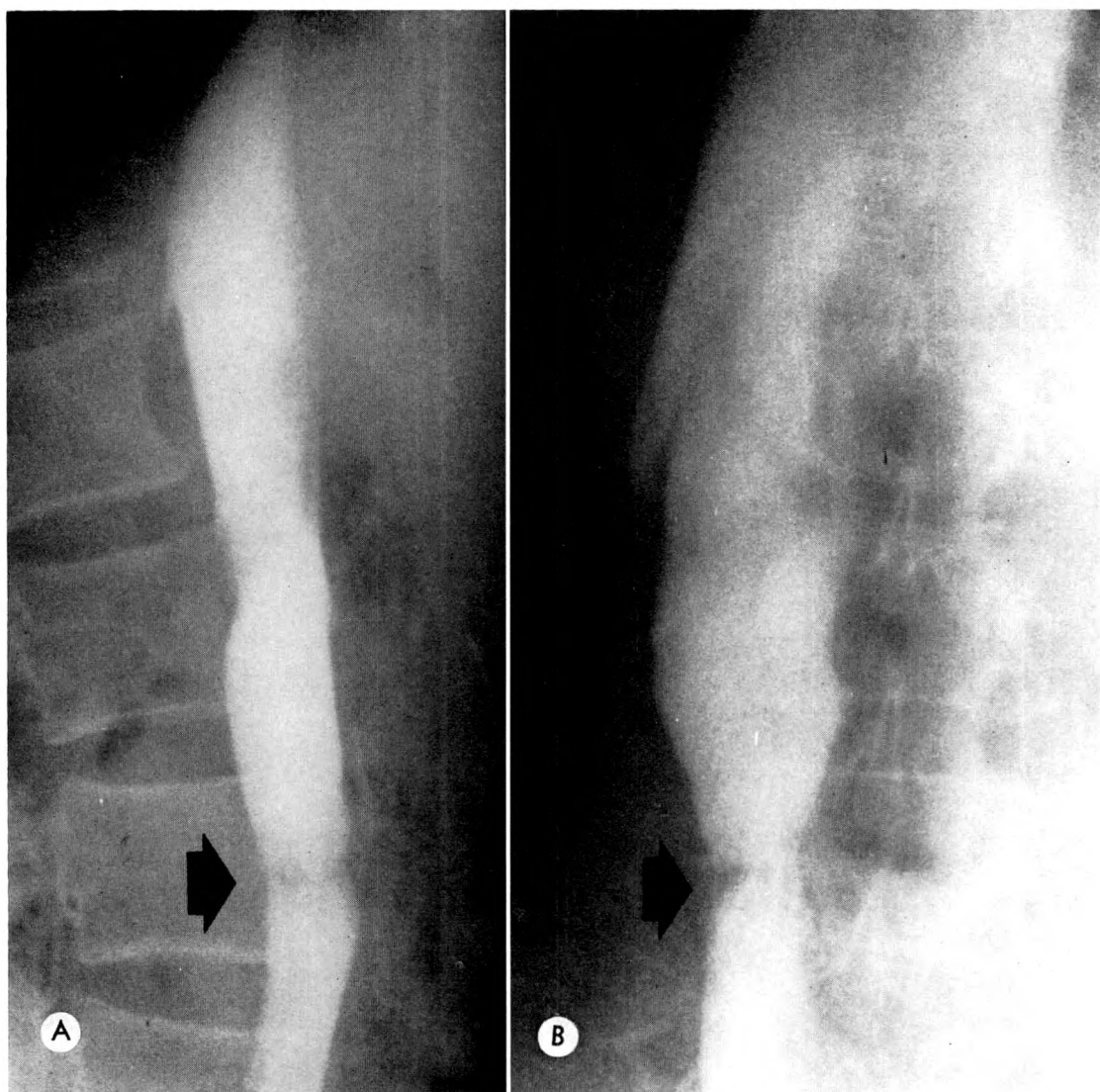


FIG. 3. (*A* and *B*) Functioning vena cava plication with partial blockage due to trapped thrombus (arrows).

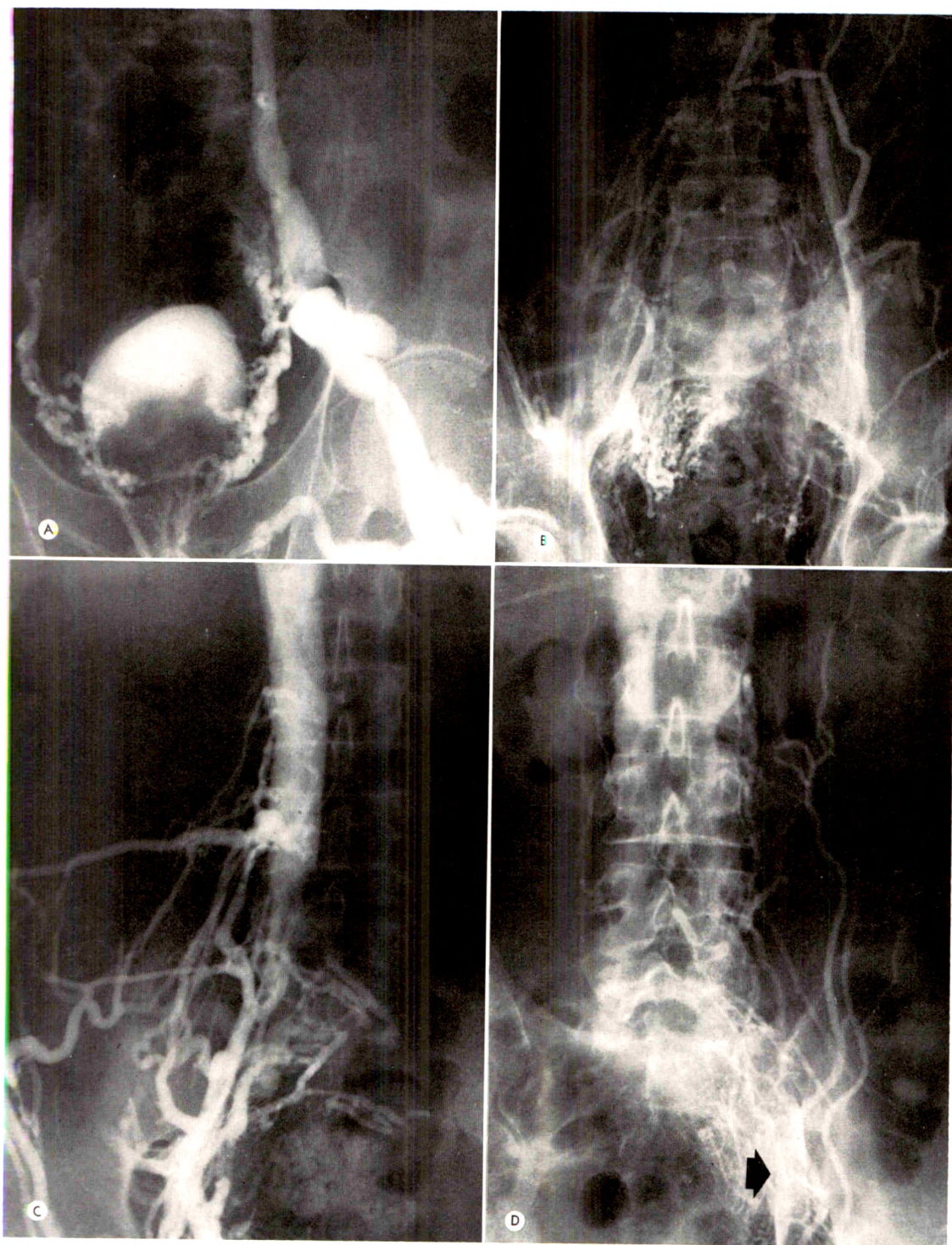


FIG. 4 (A) Complete thrombosis of plication with filling of ascending lumbar vein. Note crossover to the opposite side via perivesical and prostatic plexus. (B) Collateral flow past thrombosed plication via inferior epigastric and extensive retroperitoneal venous channels. (C) Reconstitution above thrombosed plication via deep circumflex veins. (D) Inferior epigastric collateral drainage. Note thrombus in external iliac vein (arrow).



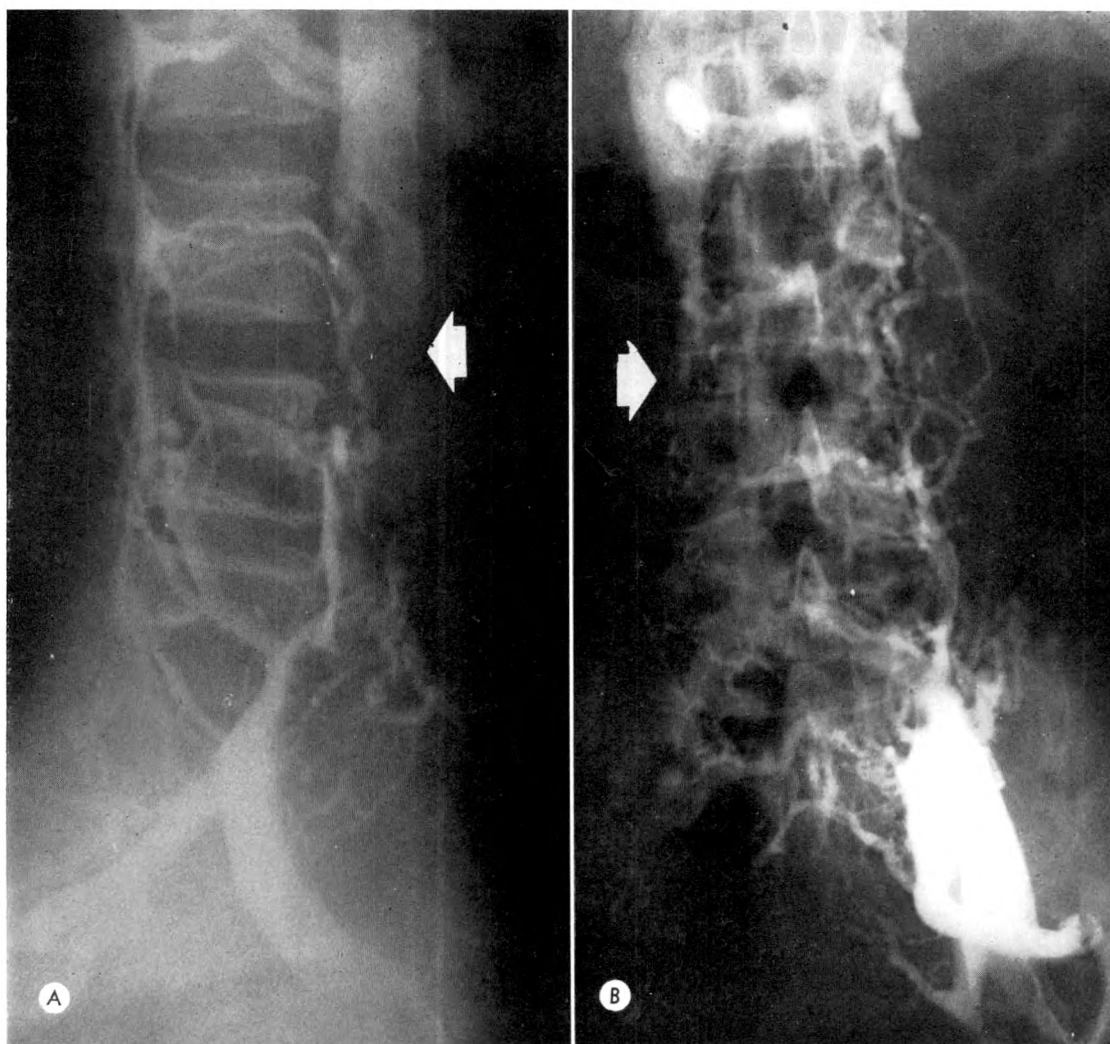


FIG. 5. (*A* and *B*) Complete thrombosis of plication site (arrows) with reconstitution above by means of vertebral veins.

obstruction, or collateral formation. Despite this appearance, the patient had a severe post-phlebitic syndrome with edema, ulceration and varicosities.

Fatal post-plication pulmonary embolism occurred in 1 case previously reported.<sup>10</sup> The patency of the plication was demonstrated by vena cavography (Fig. 9). Subsequent to this, recurrent embolization occurred and vena cava ligation was required. This also proved ineffectual. At autopsy, adherent mural thrombi were found in the atria and also in the vena cava above the operative site. Additionally, thrombi were present in the ovarian vein.

#### CLINICAL CORRELATION

Of the 22 patients examined from 6 days to 3½ years after operation, 12 were found to have patent venae cavae with little or no collateralization. Ten of the 22 cases were shown to have complete thrombosis of the plication site postoperatively, and all of these showed extensive collateralization, similar to that which has been noted following vena cava ligation.

In 4 of the 12 patients in whom patency was demonstrated, multiple vena cavograms were made up to 3 years after plication and patency persisted in all. Follow-up of thrombosed venae cavae was poor, with

the latest postoperative cavography being performed 4 months after surgery. Many of the patients who had thrombosis showed progressive diminution of their venous insufficiency symptoms; this suggested that collateral drainage improved or that recanalization occurred.

Clinical appraisal of the existence of pre- and postoperative phlebitis, edema, varicosities, and ulceration was made in the 22 cases (Table 1). As might be expected, there were more patients significantly symptomatic postoperatively (14 out of 22) than were symptomatic preoperatively (9 out of 22). Unexpectedly, there was little clear-cut difference in the clinical appearance of patients with thrombosis from those with postoperative patency. Six patients in whom the plication was patent had postoperative symptoms thought to be on the basis of obstruction of the vena cava,

and 2 patients who had roentgenographic evidence of thrombosis were asymptomatic. Thus, evaluation of the postoperative status of the vena cava on the basis of clinical findings was incorrect in 8 of the 22 cases.

Only 2 patients showed over-all improvement in the clinical status of their lower extremities postoperatively (one of these had thrombosis of the plication site). This improvement may be a reflection of better care.

Generally, there is a positive correlation between preoperative phlebitis and the postoperative phlebotic syndrome but not with thrombosis of the plication site.

There was evidence of pulmonary embolization postoperatively in 7 of the 22 cases (Table II). This includes 1 fatality from pulmonary embolization which occurred despite plication and ligation. In 6

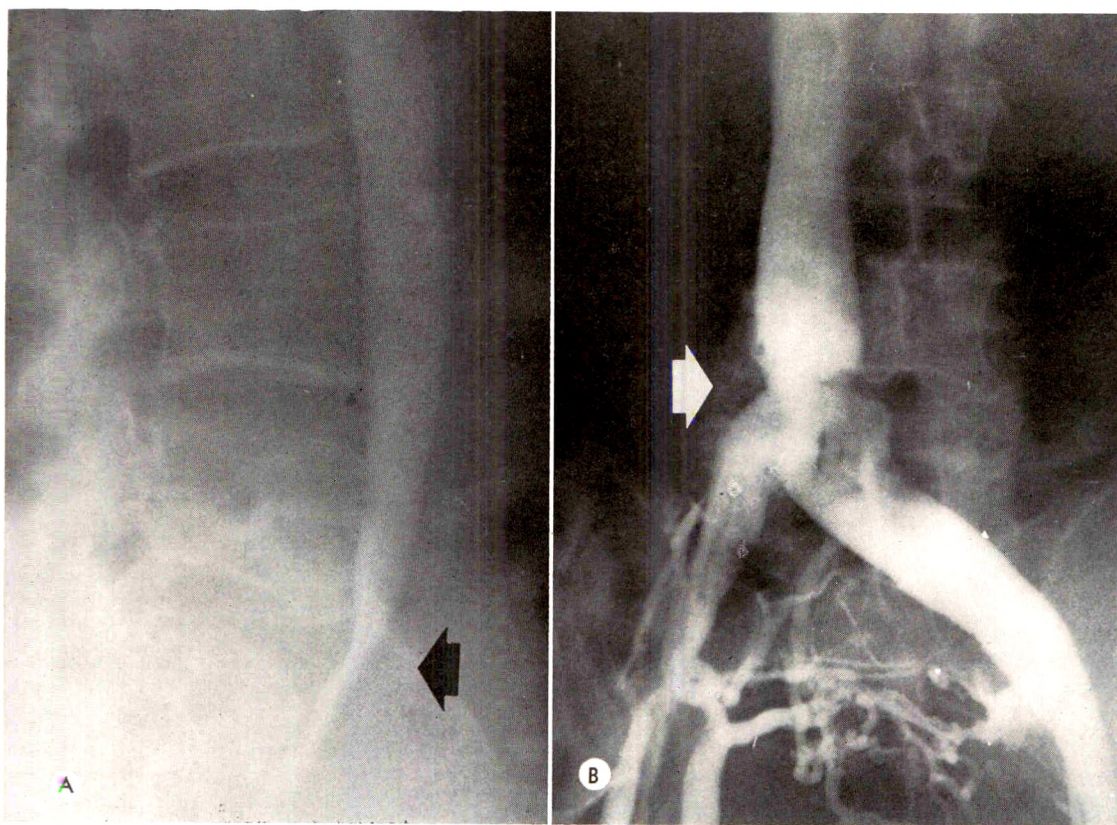


FIG. 6. (A and B) Apparent breakdown of plication site in a patient who had subsequent pulmonary emboli. A single large channel is seen in the anteroposterior projection. There is slight constriction in the lateral view (arrow).



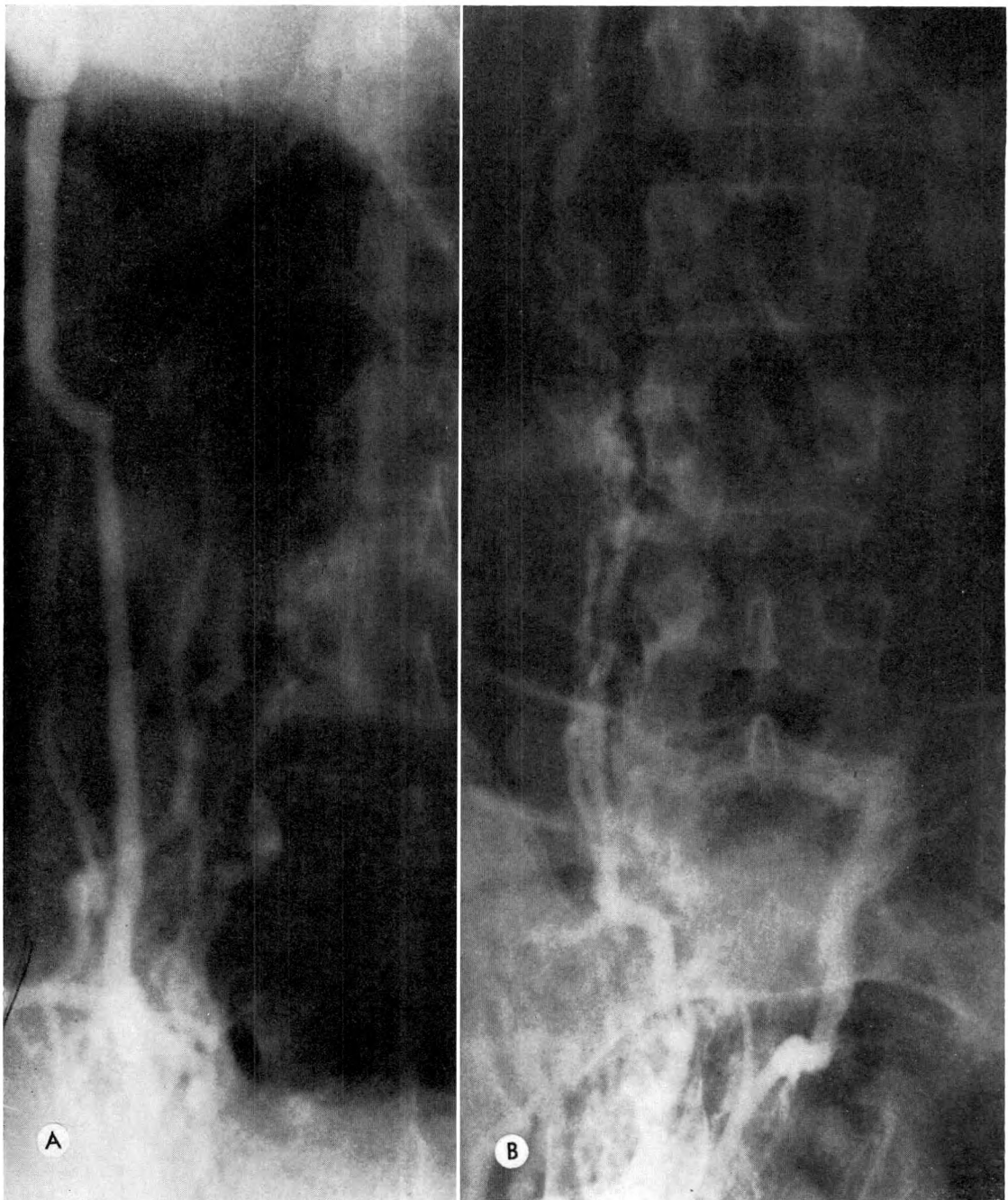


FIG. 7. (*A* and *B*) Two patients with complete post-plication caval obstruction in which clinical symptoms were mild or absent despite poor collateral formation. *A* shows major collateralization by inferior epigastric vein, *B* by ovarian veins.

of the 7 cases in which pulmonary embolization was suspected postoperatively, the post-plication vena cavogram showed patency. Only 1 patient with evidence of recurrent embolization had a thrombosed plication. This patient also had renal fail-

ure, and a diagnosis of renal vein thrombosis was entertained which would suggest that emboli originated from above the plication site. Some form of cardiac disease was present in 3 of the patients with recurrent embolization and the possibility of

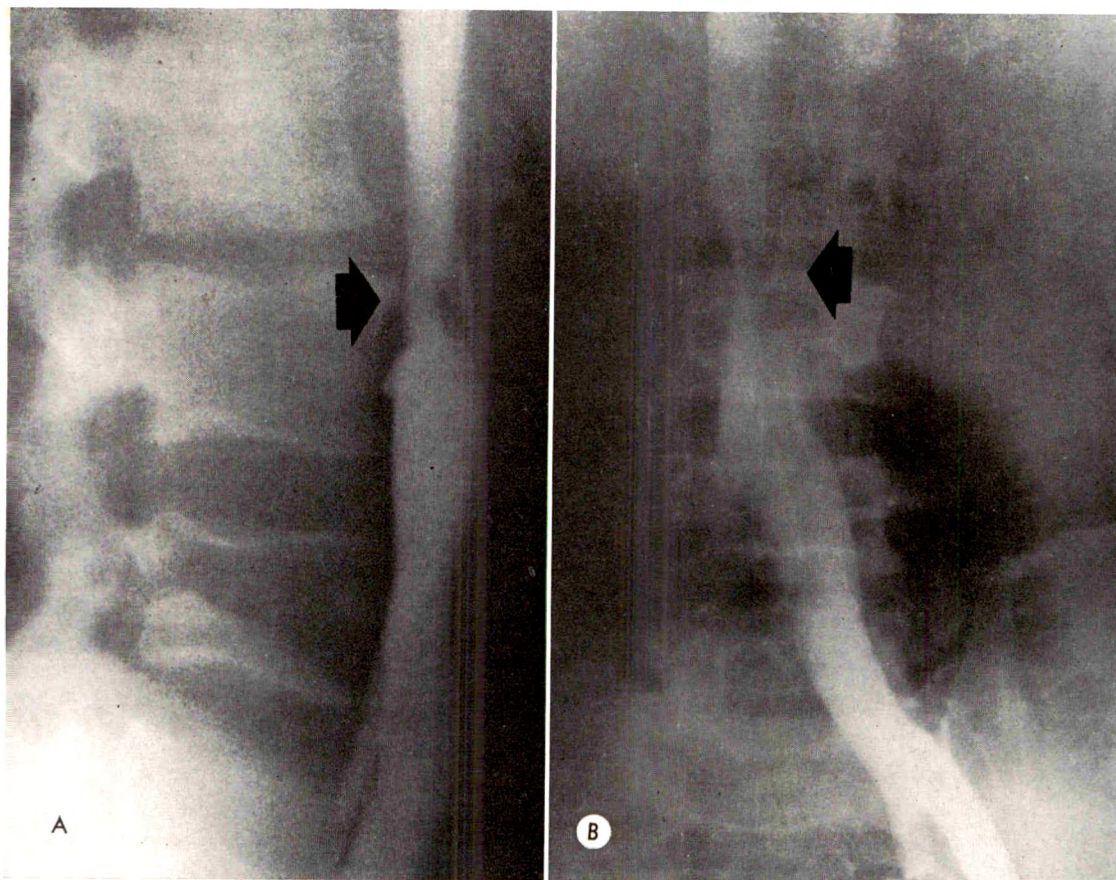


FIG. 8. (A and B) Functioning patent plication (arrows) in a patient with severe post-phlebitic syndrome. Note the lack of collateral vessels.

thrombi originating above the plication site is also suggested in these patients.

#### CONCLUSIONS

Bergan and his co-workers<sup>1</sup> have pointed out the fallacy of attempting to evaluate the postoperative plication without vena cavography and it is apparent that clinical evaluation alone is not valid in the postoperative assessment of caval patency. Nearly half of the cases in this series were shown to have thrombosis of the vena cava following plication and the procedure was, therefore, essentially a ligation.

Contrary to some reports, post-plication pulmonary emboli do occur in a substantial percentage of cases. This complication may not be a valid criticism of the plication procedures, since recurrent emboli may arise from sites other than at or below the plica-

tion site and they also have been reported following ligation.

There is a general correlation between preoperative phlebitis and the post-phlebotic syndrome, but poor correlation with the patency of the plication.

Plications that were patent in the immediate postoperative period remained so on repeat vena cavography up to 3½ years later. Late follow-up of a thrombosed plication was performed on only 1 patient in this series. We cannot state whether clinical improvement that occurred in the late follow-up period was due to new collaterals or to recanalization of the plication site.

#### SUMMARY

Vena cavographies were performed on 22 patients following compartmentalization of the inferior vena cava. Various roentgeno-





FIG. 9. Functioning patent vena cava plication (arrow) in a patient who developed recurrent emboli. Despite a subsequent caval ligation, the patient died of embolization, probably from the ovarian vein.

graphic appearances of the vena cava following plication were demonstrated. Twelve of the patients examined were

found to have patency of the plication site, whereas 10 were found to have complete thrombosis following the surgical procedure. There was poor correlation between clinical and roentgenographic findings, and accurate clinical assessment of the status of the vena cava could not be established prior to the performance of vena cavography.

Evidence of pulmonary embolization was present postoperatively in 7 of the 22 cases and these included 1 case of fatal pulmonary embolus which occurred after ligation of the vena cava. Six of the postoperative pulmonary embolization patients were found to have patency of the plication, and in 2 there was suggestion of partial or complete breakdown of the plication site.

Plication of the vena cava does not appear to be the final solution as regards the problem of pulmonary embolization.

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TABLE I  
CLINICAL EVALUATION OF 22 POST-PLICATION CASES

No. of Cases	Roentgenologic Status	Lower Extremity Preoperative Symptoms		Lower Extremity Postoperative Symptoms	
		Absent	Present	Absent	Present
10	Thrombosed	5	5 (4 mild, 1 severe)	2	8 (3 mild, 5 severe)
12	Patent	8	4 (3 mild, 1 severe)	6	6 (3 mild, 3 severe)
22		13	9	8	14

TABLE II  
BASIS FOR DIAGNOSIS OF POST-PPLICATION PULMONARY EMBOLIZATION: 7 OF 22 CASES

Pa- tient	Chest Roentgeno- gram	Electro- cardiogram	Hemop- tysis	Chest Pain	Vena Cavogram	Other Evidence	Other Diseases
1	+	+	+	+	Patent	Ligation Autopsy	Hypothyroid
2	+	—	+	+	Patent	+Scan	Mitral Disease Heart Failure Atrial Fibrillation
3	+	—	+	+	Patent (2 studies) Breakdown		Diabetes
4	+	+	+	+	Thrombosed	Renal Failure	—
5	+	+	—	—	Patent (3 studies)	? Positive Pulmonary Arteriogram	Asthma Heart Failure
6	+	—	—	—	Patent	—	Diabetes Heart Failure Myocardial Infarction
7	—	—	—	+	Patent (2 studies) Partial Breakdown	—	Neurotic

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## ARTERIOGRAPHY IN CIRCUMFERENTIAL COMPRESSION EDEMA

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SEVERE crushing injury to an extremity produces marked soft tissue swelling with edema and ecchymosis. This may interfere with accurate clinical evaluation of the circulatory status and may raise serious questions relative to the proper management of the patient.

The importance of arteriography in the evaluation of acute arterial injuries has been cited frequently,<sup>1,7,10</sup> although occasionally discredited.<sup>8,9</sup> Others stress the usefulness of arteriography for the evaluation of patency at the time of arterial surgery.<sup>3</sup>

The value of carefully planned arteriography, individually tailored to best fit the problem at hand, is emphasized by the following case report.

### REPORT OF A CASE

A 40 year old male was admitted to Ohio Valley General Hospital, November 12, 1965, with a severe crushing injury to his right shoulder and arm. His extremity had been "pinned" between an automobile chassis and the ground for a period of 6 hours. A co-worker was crushed to death in the same accident.

Physical examination disclosed the left shoulder and arm to be extremely swollen and ecchymotic with board-like induration (Fig. 1). No arterial pulsations could be palpated. However, the patient's blood pressure and pulse rate were normal and the extremity, although incapable of movement, exhibited only slightly reduced skin temperature. Roentgenograms showed no evidence of fracture.

Arteriography was performed. A catheter was introduced into the right femoral artery and cautiously passed up the aorta into the distal left subclavian artery. From this position, rapid serial film angiography was performed over a 9 second period, which indicated complete circumferential obstruction of all visible arterial flow at the mid brachial level (Fig. 2, *A* and *B*). Even delayed arteriograms at the 9 second interval indicated a sharp line of demarcation produced by muscular arterial branches at the

obstruction level with gradual minimal advancing opacification of the brachial artery.

Following arteriography, moderate warmth was maintained in this patient's pulseless extremity, and his fingernail beds, although ischemic in appearance, showed a faint pinkish color. There were no clinical signs of significant venous occlusion. For these reasons, it was determined to manage this case with nonsurgical means including elevation of the extremity, anticoagulants and varidase. The edema of the extremity gradually disappeared and pulses became palpable on the sixth day following the injury.

Direct percutaneous axillary arteriography was performed after return of the extremity pulses, (Fig 3, *A* and *B*) which indicated completely normal findings with prompt opacification of the arterial tree on serial film studies and normal venous circulation of the extremity. At the time of this writing (7 weeks after injury), the patient is gradually recovering motor and sensory deficit, confined to the lower forearm and hand.

### COMMENT

A crushing injury of the extremity may traumatically interfere with its arterial circulation by various mechanisms. These



FIG. 1. Severely edematous and ecchymotic shoulder and arm with absent peripheral pulses due to a crushing injury.



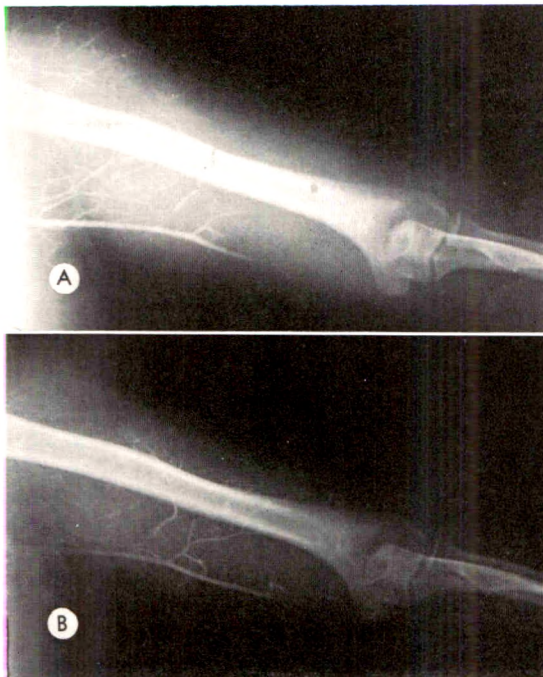


FIG. 2. Transaortic left axillary catheter arteriography via right femoral artery route was performed a short time after injury. (A) One and one-half second interval arteriogram showing occlusion of arterial flow at mid brachial artery level. (B) Nine second interval arteriogram indicating circumferential compression of all visible arterial flow at the level of the mid brachium. A transverse zone of demarcation is delineated by small muscular arterial branches where opacification is abruptly terminated.

mechanisms include: 1. compression of vessels by subfascial hematoma and edema.<sup>5</sup> Evidently, this was the cause of arterial obstruction in this case report; 2. subintimal arterial hemorrhage;<sup>7,10</sup> 3. intimal arterial tear with secondary thrombosis;<sup>7</sup> 4. incomplete division of an artery with simple blood loss, false aneurysm or arteriovenous fistula;<sup>1,7</sup> 5. complete arterial transection;<sup>10</sup> 6. arterial obstruction due to entrapment of the artery between fracture fragments in an extremity fracture;<sup>5,10</sup> 7. massive venous occlusion with secondary arterial inflow obstruction;<sup>4,10</sup> and 8. spasm.<sup>3,7</sup>

Thus, with many potential mechanisms of ischemia due to extremity trauma, the value of safe and efficient arteriography, performed early, seems obvious.

The hazard of embarrassing an already

compromised circulation related to extremity trauma can be avoided if approach to the artery in question is by entrance to the arterial tree from a distant normal artery, using a transaortic catheter approach to the extremity artery under study.<sup>2</sup>

Rapid serial arteriograms, procured over a relatively long time interval (10 to 15 seconds), will provide reliable information relative to circulatory dynamics. Moreover, such serial studies plus "flooding" of the

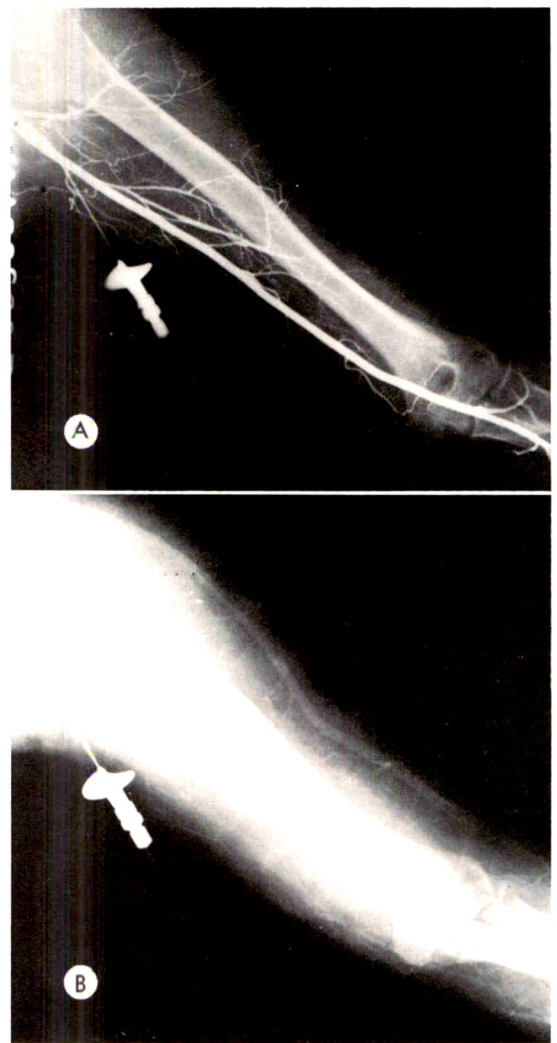


FIG. 3. Repeat axillary-brachial arteriography was performed by direct axillary needle puncture 1 week after initial injury following subsidence of edema and return of extremity pulses. Serial angiograms at (A) 1 second and (B) 6 second intervals show return of normal arterial and venous flow.



artery with adequate amounts of contrast material should avoid a possible error of interpretation, which may be produced by an unopacified column of blood proximal to a point of obstruction. This has been emphasized by Logan and Goudelock.<sup>6</sup>

In this case, clinical progress and serial angiography strongly indicate that an effective microcirculation to an extremity can exist in spite of failure to arteriographically demonstrate arterial blood flow beyond an obstruction level.

#### SUMMARY AND CONCLUSIONS

1. This case emphasizes the potential value of catheter arteriography and rapid serial filming in the management of acute traumatic pulseless disease of an extremity.

2. Arteriographic failure to opacify visible arterial flow to the periphery of a swollen, traumatized extremity does not exclude a physiologically effective microcirculation.

3. Although clinical judgment may be paramount in the management of extremity trauma with ischemia, transaortic catheter arteriography may provide valuable information with relative safety and simplicity.

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## STATIONARY WAVES OF THE SUPERFICIAL FEMORAL ARTERIES\*

### REPORT OF A CASE OCCURRING IN A PATIENT WITH RHEUMATIC HEART DISEASE AND POPLITEAL ARTERIAL EMBOLI

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THEANDER,<sup>19</sup> in 1960, was the first to suggest that the rarely occurring regularly transverse striations seen in the contrast column in an artery following arteriography was due to stationary arterial waves. Hitherto, following the report of Wickbom and Bartley,<sup>21</sup> it had been attributed to vascular spasm and needless dosages of intravascular antispasmodic agents were prescribed for treatment of the "spasm." In 1964, Mayall,<sup>8</sup> using dilute contrast medium and water in a tube, postulated that the phenomenon was due to rippling of the interface between the upper layer of blood and lower layer of contrast medium, when slipping occurred between the two layers owing to different rates of flow. Two years later, by ingeniously substituting turpentine for blood, Mayall<sup>9</sup> was able to show that after a slight tilt, the heavier contrast medium ran beneath the upper layer towards the end of a sealed tube, but the interface remained smooth. A sharper angle of tilt produced more rapid flow with rippling of the surfaces. These ripples increased both in wavelength and amplitude when the experiment was repeated with increasing angles of tilt. Eventually, a markedly rapid tilt caused turbulence, sufficient to mix the two layers and destroy the waves. Narrower tubes produced the same results, but the waves were of smaller amplitude and wavelength. Thus, the observation of Theander<sup>19</sup> that the waves were of smaller amplitude and shorter wavelength in smaller arteries was confirmed.

Stationary arterial waves have been re-

ported in the peripheral vascular system,<sup>4,8,18,21</sup> and in the mesenteric,<sup>8,22</sup> renal,<sup>18,22</sup> and internal carotid<sup>14</sup> arteries. In the patient herein reported, the superficial femoral arteries contained stationary waves following arteriography which also demonstrated bilateral occlusion of the popliteal arteries from emboli arising from the left atrium of a woman with valvular disease due to rheumatic heart disease. This communication re-emphasizes the physical and harmless nature of stationary waves and disposes of the "enigma"<sup>4</sup> that they have created.

#### REPORT OF A CASE

A 29 year old Caucasian housewife (N.Y.H. No. 908408) was admitted on April 8, 1962, with the complaint of left flank pain of 1 day's duration. She was said to have had chorea at the age of 6 years, which recurred at the age of 12 years and was severe enough to require hospitalization. She was asymptomatic during the early weeks of her only pregnancy, at the age of 24 years, but was digitalized and spent most of the time at bed rest prior to delivery. A year later, she had improved so much that digitalis was discontinued. At the age of 27 years, albuminuria, dyspnea on exertion, fatigability, and atrial fibrillation developed and redigitalization was necessary. For 4 months prior to admission, increasing dyspnea and edema of the extremities occurred and diuretics were added to the treatment. One day before admission, she experienced a severe, sharp left flank pain which did not radiate but was accompanied by numbness and tingling of both feet.

Physical examination revealed a well developed and well nourished woman in moderate

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distress. The lungs were clear. The heart was enlarged and the left border of the cardiac dullness extended to the sixth left interspace in the anterior axillary line. There was a prominent left ventricular heave. The pulmonic second sound was loud and split. Atrial fibrillation at the rate of 88 was present at the apex of the heart. An opening snap was heard along the left sternal border at the third and fourth interspaces. A holosystolic murmur (Grade IV) was present at the apex of the heart which radiated into the axilla and along the left sternal border. An early (Grade II to IV) systolic murmur was heard along the left sternal border and increased with respiration. A diastole rumble was also heard along the left sternal border. There were no murmurs at the base of the heart or in the neck. The blood pressure was 100/60 mm. Hg. The liver was enlarged, the abdomen was soft, and the left flank was tender. There was no edema of the legs but both popliteal arteries had diminished pulsations.

Laboratory data showed an increased leukocyte count (15,000) with 87 per cent polymorphonuclear cells. The urine contained a large amount of albumin. The electrocardiogram showed atrial fibrillation and digitalization effects. Roentgenographic studies of the chest showed enlargement of the heart, especially of the atria and right ventricle and congestive changes at the right base (Fig. 1). Angiocardiography on April 12, 1962, demonstrated tremendous right atrial dilatation; the right ventricle, pulmonary artery, and left atrium were enlarged (Fig. 2*A*). The latter measured 15.5 by 9 cm. (average normal=8.5 by 5 cm.<sup>6</sup>). Left atrial thrombi, measuring 2 by 2 cm. in the vicinity of the atrial appendage and 3 by 3 cm. in the region of the mitral valve, were evident (Fig. 2*B*). Percutaneous trans-femoral aortograms, made 2 days later, demonstrated a thrombus of a branch of the left renal artery (Fig. 3, *A* and *B*). Serial studies revealed unopacified filling defects of tricuspid insufficiency<sup>2</sup> with delay in emptying and variation of the radiopacity between the right atrium and ventricle, consistent with tricuspid stenosis.<sup>20</sup> The peripheral vascular system exhibited stationary waves in the superficial femoral arteries (Fig. 4*A*) and occlusion of both popliteal arteries (Fig. 4*B*).

The patient was treated with anticoagulants and improved. She was discharged on May 11, 1962, and subsequently visited the anticoagulant and cardiac clinics regularly. She was re-

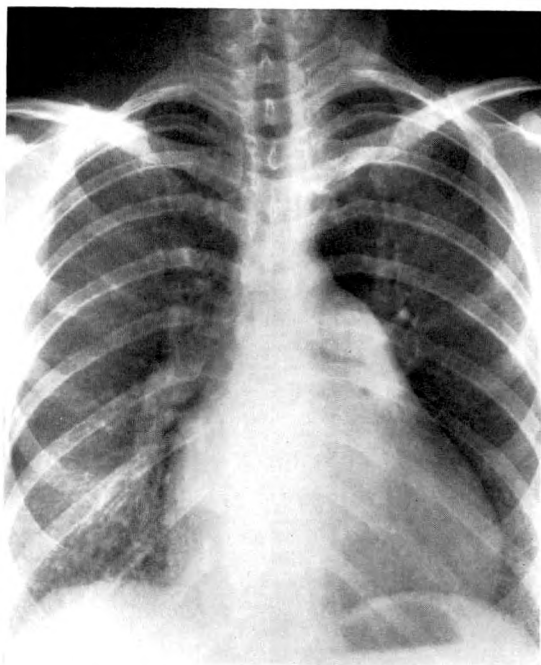


FIG. 1. Conventional frontal teleroentgenogram of the chest showing the markedly enlarged heart, pulmonary artery and branches, and congestive hilar and basilar lung changes.

admitted on June 11, 1963, because of sudden onset of pain in both legs of 1 hour's duration. She had been on a long automobile journey over the weekend, had slept well, but while eating breakfast developed leg numbness and pain. She was seen in the emergency room and a saddle embolus at the bifurcation of the aorta was suspected because of the sudden onset of pain and numbness, poor peripheral pulses, and coldness of the feet. While being wheeled into the operating room for exploratory study of the peripheral vascular system, she developed severe dyspnea. Examination disclosed diffuse rales all over the chest with pulmonary edema. Accordingly, she was transferred to the medical ward where phlebotomy, intermittent positive pressure respiration, and diuretics relieved her. Subsequently, the extremities became warm and their pulsations improved. Recovery continued and she was discharged on June 28, 1963.

She was readmitted on November 16, 1963, and 3 days later, mitral commissurotomy, using open-heart technique with a rotating disk type pump oxygenator, was performed. The left atrial appendage was found to be completely filled with soft clots. The mitral valve orifice

measured only 0.5 cm.<sup>2</sup>. A Gerbode dilator was used to stretch the mitral valve to about 3.0 cm.<sup>2</sup>. The patient did well postoperatively, and prior to discharge, atrial fibrillation was converted to normal sinus rhythm by electrical shock, and she was discharged on December 14, 1963. Anticoagulant therapy was continued.

She was readmitted on April 12, 1964, for direct current conversion of atrial fibrillation. This returned to regular sinus rhythm with occasional atrial premature contractions. The patient has continued to improve. The cardiac rhythm was regular until October 8, 1964, when it again became totally irregular (atrial fibrillation). The cardiac silhouette was unchanged, and on her last visit to the clinic on February 11, 1965, her only complaint was tiredness in the legs.

#### DISCUSSION

Angiocardiography has provided the means of visualizing the heart in rheumatic heart disease.<sup>1,2,6,13,17</sup> Left atrial thrombi and tumors have also been differentiated with this technique.<sup>7,15,16</sup> Selective pulmonary arterial and left atrial injections of contrast media for detection of thrombi have recently come into greater use.<sup>3,11,12</sup> Figure 2*B* illustrates the atrial thrombi visualized with the intravenous method of angiocardiography. Opacification of the en-

tire cardiovascular system also permitted the diagnosis of a huge right atrium due to tricuspid regurgitation<sup>2</sup> and stenosis (Fig. 2*A*).<sup>20</sup> Angiocardiographic diagnosis of left atrial thrombi and tight mitral stenosis was verified by open-heart surgery.

Figure 3*A* shows the angiographic appearance of the left renal arterial embolus that occurred prior to the first admission of the patient. The radiolucent filling defect is characteristic of a renal thrombus and appears similar to that described by Morrow and Amplatz,<sup>10</sup> except, of course, for the fact that the one demonstrated above occurred spontaneously and did not complicate the arteriographic study. Surgical exploration of thrombotic renal arterial lesions is just becoming routine,<sup>5,10</sup> and it is fair to predict that surgery rather than anticoagulant therapy alone will be the treatment of choice. This is logical, for removal of the thrombus may be expected to preserve renal function, especially when the renal embolus is of recent origin. Figure 3*B* shows the widespread renal infarction that followed the emboli.

Stationary arterial waves have been attributed to arterial spasm and intra-arterial instillation of priscoline has been advocated

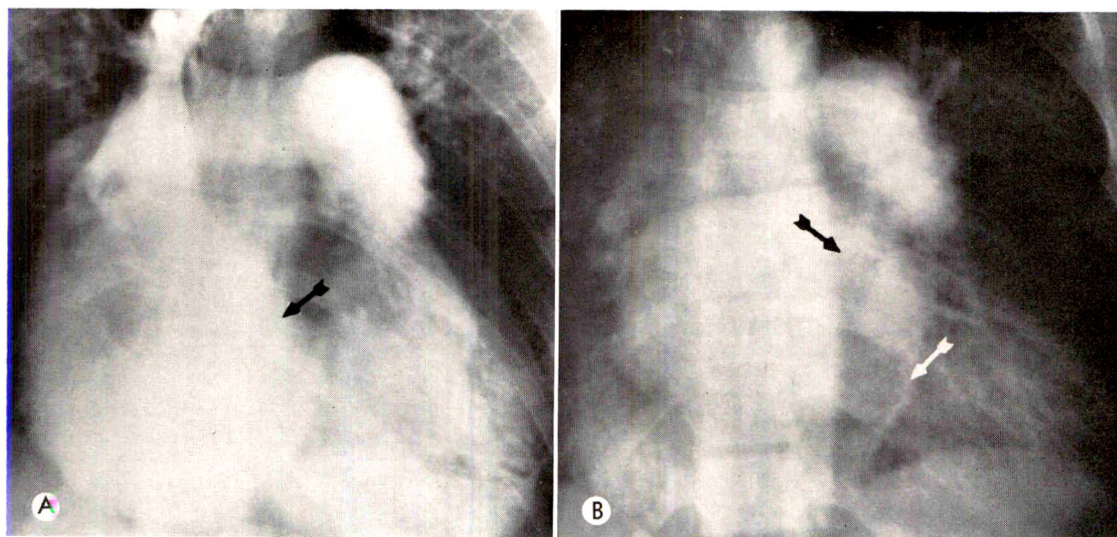


FIG. 2. (A) Frontal intravenous angiocardiogram showing the huge right atrium (arrow), enlarged right ventricle, pulmonary artery, and branches. (B) Left heart angiocardiogram showing thrombus in the region of the left atrial appendage (black arrow) and at the base of the atrium above the mitral valve (white arrow).



for the relief of the spasm.<sup>21</sup> However, since the stationary waves do not produce ill effects, treatment is not indicated. The transverse striations have been likened to a bamboo or a string of pearls,<sup>8</sup> and called "corrugated arteries."<sup>4</sup> Following Wickbom

and Bartley's<sup>21</sup> initial paper, which apparently described only a single case of stationary waves during peripheral arteriography, Theander<sup>19</sup> reported 4 cases among 160 patients who had peripheral arteriography. As in our case, 2 followed pe-

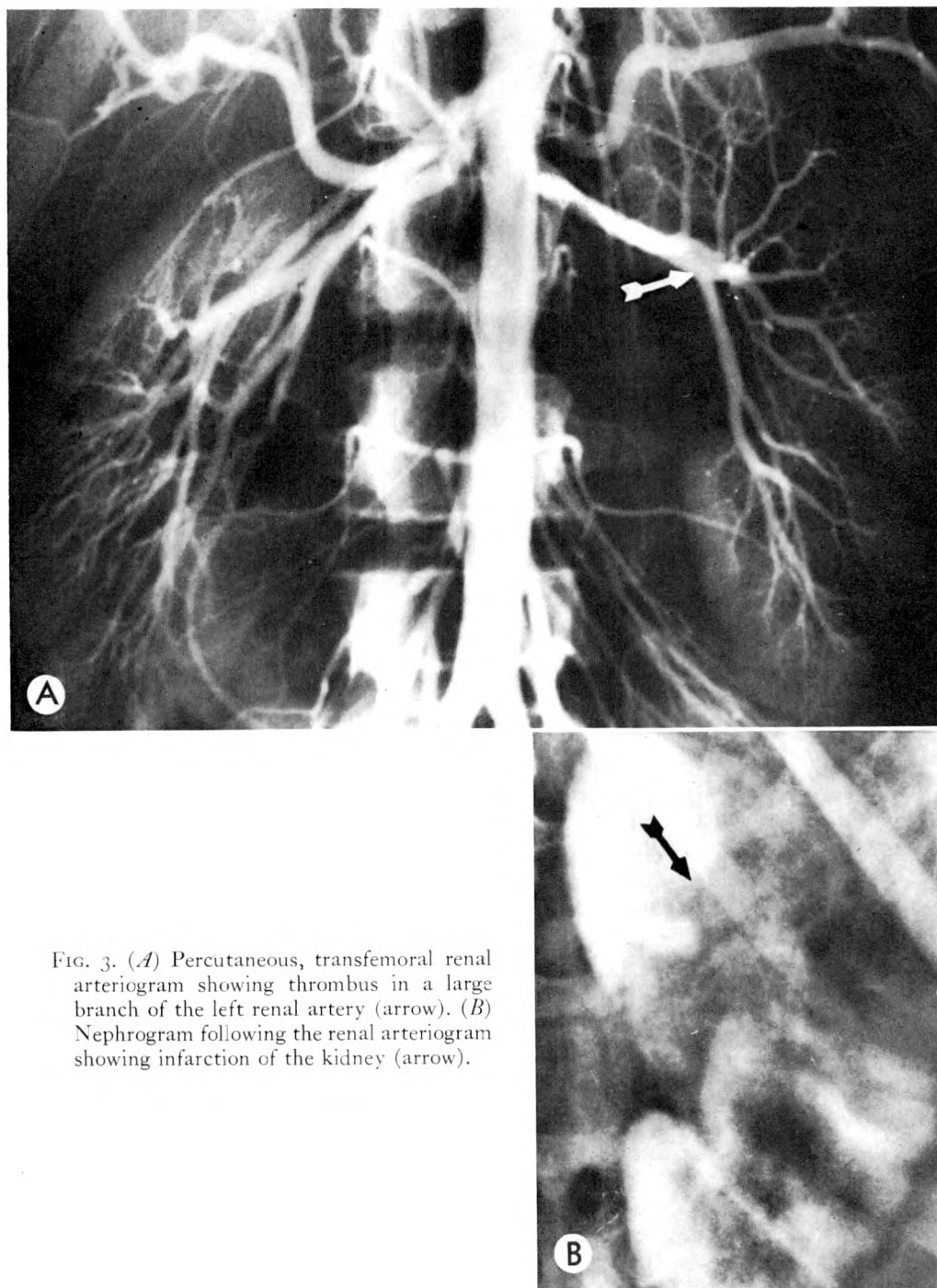


FIG. 3. (A) Percutaneous, transfemoral renal arteriogram showing thrombus in a large branch of the left renal artery (arrow). (B) Nephrogram following the renal arteriogram showing infarction of the kidney (arrow).

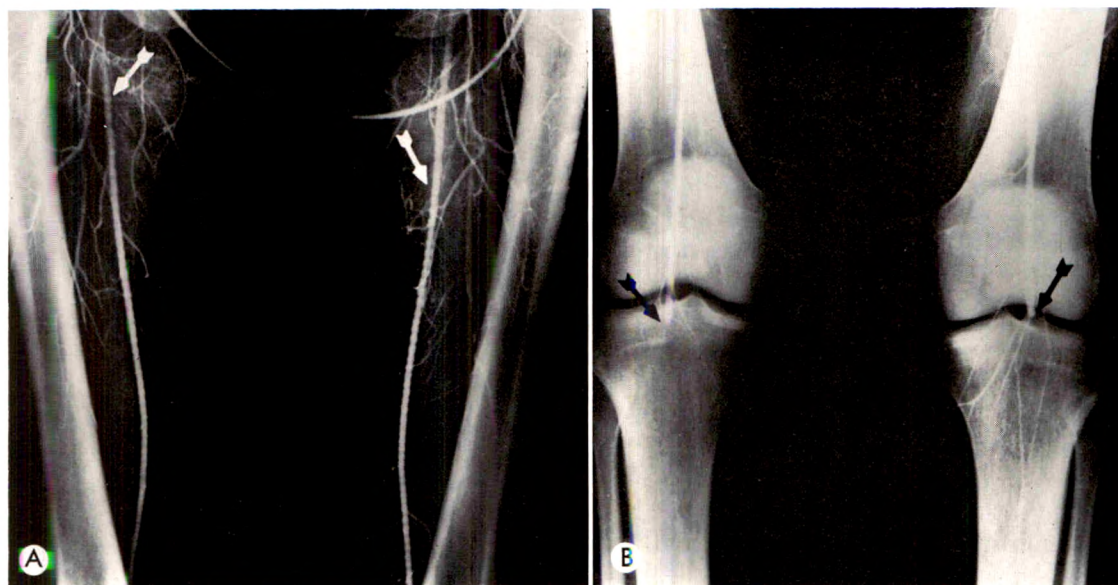


FIG. 4. (A) Peripheral arteriogram showing bilateral stationary arterial waves in the superficial femoral arteries (arrows). (B) Serial arteriogram showing the bilateral occlusions of the popliteal arteries (arrows).

ipheral vascular emboli in men suffering from mitral stenosis. The third was in a man with Buerger's disease, while the last had vascular lesions of unknown etiology. Three other patients with arterial embolism in Theander's series had arterial emboli associated with advanced arteriosclerosis, but an adequate collateral circulation was present and apparently prevented stationary waves. He, therefore, rightly concluded that stationary arterial waves occurred only when flow of the blood and contrast material is impeded. Sutton<sup>18</sup> has described the visualization of stationary arterial waves in several femoral arteriograms, many of which were in patients with Buerger's disease. According to Mayall,<sup>8</sup> he later reported single examples in the mesenteric, iliac, renal, and internal carotid arteries. Foster and his colleagues<sup>4</sup> found an incidence of 7 patients with "corrugated" arteries in over 600 patients having arteriography. None had thromboangiitis.

Although Foster and associates<sup>4</sup> question Mayall's theory of interface disturbance being the explanation for stationary arterial waves, Mayall's experimental proof seems rational.<sup>9</sup> It would appear that stationary arterial waves form only when the difference of velocity between the two layers,

blood and contrast medium, is sufficient to generate ripples but not enough to produce mixing. He explains the uncommonness of the condition by stating that the critical situations producing variation in velocity of the two liquids are rare.<sup>9</sup> Mayall also attributes the striated "string of pearls" pattern to the action of the contrast medium which at the crests of the waves is deeper than the troughs. He recommends that simultaneous frontal and lateral roentgenograms be made of stationary arterial waves. He predicts that in lateral view the lower margin of the contrast layer will be a smooth line following the arterial wall, while the upper margin will be wavy.<sup>9</sup>

#### SUMMARY AND CONCLUSIONS

Stationary arterial waves were demonstrated in the superficial femoral arteries of a 29 year old woman following emboli to the left renal and both popliteal arteries. Angiocardiography showed that left atrial thrombi due to advanced rheumatic valvular heart disease were the cause of these. Anticoagulant treatment was prescribed. Despite this, further embolization, cardiac arrhythmia, and heart failure recurred, requiring open-heart surgery for the mitral stenosis and removal of the atrial thrombi.



She was improved, although electrical conversion of atrial fibrillation was subsequently needed.

The case is chiefly of interest because of the stationary arterial waves. These have also been reported in the peripheral vascular, visceral, and extracranial arteries. A mechanical explanation for these was proposed by Theander in 1960, and this was recently confirmed by Mayall, who, with the use of a glass model, demonstrated that disturbances of the interfaces of blood and contrast medium owing to rippling and obstruction to blood flow produced stationary waves during arteriography. This theory confirms the innocent nature of the phenomenon and makes unnecessary intra-arterial medication, which was originally advocated because the stationary waves were attributed to arterial spasm. It also disposes of the "enigma" of their origin and appearance during arteriography.\*

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## FIBROMUSCULAR HYPERPLASIA\*

### AN ARTERIAL DYSPLASIA OF INCREASING CLINICAL IMPORTANCE

By A. J. PALUBINSKAS, M.D., DOROTHEE PERLOFF, M.D.,  
and THOMAS H. NEWTON, M.D.

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**F**IBROMUSCULAR hyperplasia (FMH) of the renal arteries producing renal artery stenosis and secondary hypertension is now a well recognized entity.<sup>2,3,5,8,11</sup>

Although its occurrence in extrarenal arteries was suggested without proof in 1963,<sup>4</sup> the lesion was regarded as limited to the renal arteries<sup>10</sup> until 1964. In that year a patient with microscopically proved FMH of the celiac artery was reported<sup>6</sup> and in July, 1965, the first histologically proved case of FMH of the internal carotid arteries was published.<sup>1</sup>

In the study of a large group of patients with FMH of the renal arteries seen at the University of California Medical Center, two important findings have been noted. First, the studies confirm that the lesion is not limited to the renal arteries. Secondly, in this group of patients with FMH, there appears to be an increased incidence of intracranial aneurysms.

The purpose of the authors is to call attention to these two findings and to stress the increasing clinical importance of this arterial lesion.

#### ETIOLOGY

The etiology of FMH is unknown. Although the lesion in the renal arteries has been seen in all ages and in both sexes, more than 85 per cent of the patients studied at the University of California Medical Center were females in the young adult or middle age range. The youngest patient with microscopically proved FMH of the renal arteries was a male infant of 17 months and the oldest was a female of 56 years.

#### PATHOLOGIC CHANGES

Wellington<sup>9</sup> has described the gross and histologic features of the lesion in the renal

arteries. The most common histologic pattern was found to be irregular thickening of the vessel by proliferation of smooth muscle and fibrous tissue of the media. Stenosis of the artery was produced by thickening of the vessel wall or by projection of thickened septa and ridges into the lumen.

#### ROENTGEN FEATURES

Certain characteristic angiographic signs in the roentgen diagnosis of FMH have been stressed.<sup>7</sup>

First, FMH is characteristically located in the middle and distal thirds of the main renal vessel, sometimes extending into the branches. This location is unlike most atherosclerotic stenoses of the renal artery which ordinarily occur in the proximal part of the vessel at the aortic origin.

Secondly, moderate or severe FMH lesions of the renal arteries have a distinct angiographic appearance (Fig. 1, A-D). The diseased portion of the vessel appears corrugated or pleated, resembling a string of beads of varying size. Aneurysms within the diseased segment, or separate from the lesion and located within the kidney itself, are common (Fig. 1, B and C).

#### MATERIAL

##### FMH IN THE RENAL ARTERIES

Through 1964, 70 hypertensive patients with definite roentgenographic evidence of FMH of the renal arteries have been studied at the University of California Medical Center in San Francisco. The lesion was bilateral in about half the patients.

Vascular surgical procedures were performed in 42 of the 70 patients and the FMH of the renal arteries was confirmed microscopically. Blood pressure returned to

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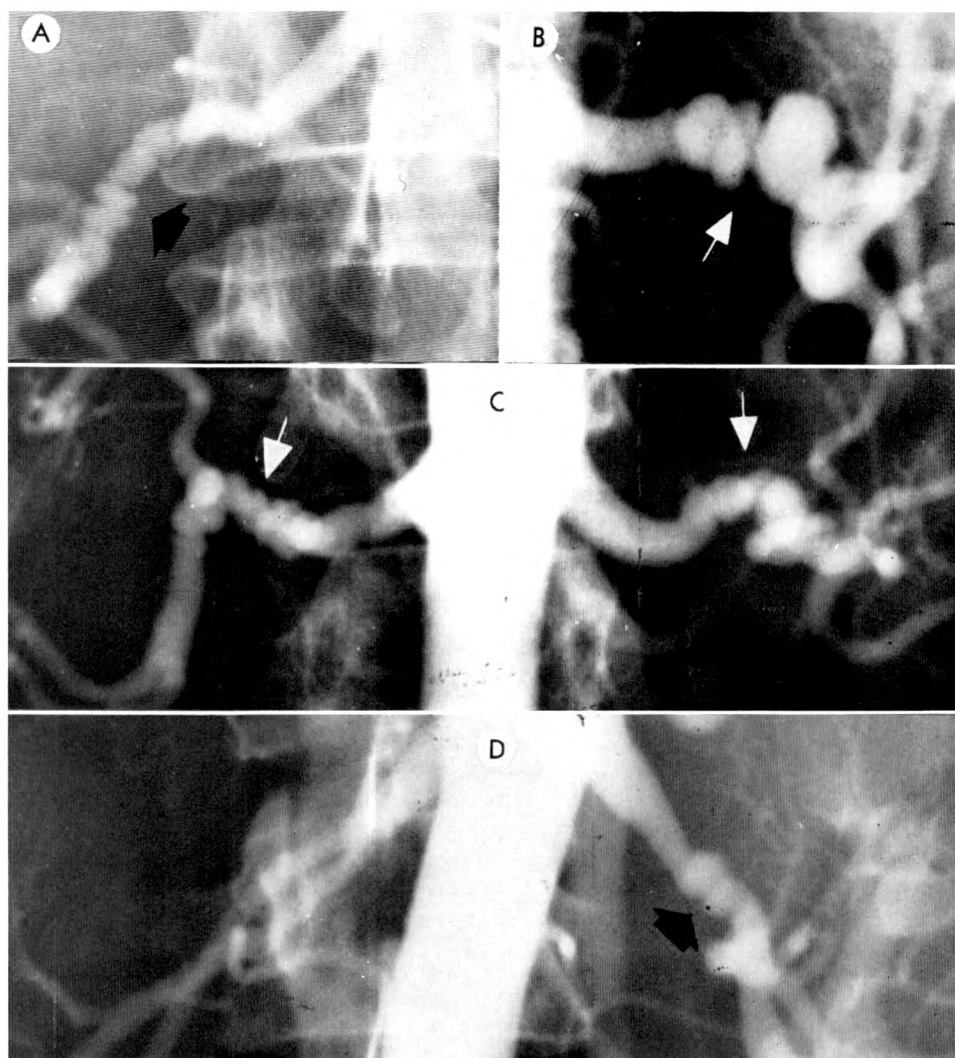


FIG. 1. (A-D) Typical arteriographic appearance of proved fibromuscular hyperplasia (arrows) of the renal arteries in 4 adult females with hypertension. (B and C) Aneurysms in the involved arteries. Note the symmetric involvement of the two renal arteries in C.

normal or was significantly reduced in about 75 per cent of the operated patients.

Figure 1, A-D demonstrates the typical appearance of proved moderate to severe FMH of the renal arteries in 4 female patients.

#### FMH IN EXTRARENAL ABDOMINAL ARTERIES

Except in rare instances, FMH lesions in nonrenal abdominal vessels are usually silent and discovered incidentally in the course of abdominal angiography performed for other reasons.<sup>6</sup>

The occurrence of proved FMH in ab-

dominal arteries other than renal is illustrated by the following case report.

**CASE 1.** A 47 year old white woman was admitted to the University of California Medical Center in December, 1963 because of headaches, blurring of vision in the right eye, and hypertension as high as 230/130 mm. Hg.

A renal arteriogram demonstrated moderately severe FMH of the right renal artery with an aneurysm arising at its proximal branches, mild FMH in two arteries to the left kidney, and changes resembling minimal FMH in the right external iliac artery (Fig. 2).

On the day following a differential kidney

function study, the patient suddenly became unresponsive, vital signs deteriorated rapidly, and cardiac arrest occurred. The cause of death was attributed to the anoxic effects of the cardiac arrest, but at postmortem examination no direct cause for the arrest was found.

Microscopic study of tissues pertinent to this discussion revealed FMH in the renal, superior mesenteric, celiac, and external iliac arteries. There were also small saccular aneurysms of the superior mesenteric and right renal arteries (Fig. 3, A and B).

#### FMH IN EXTRA-ABDOMINAL ARTERIES

In July, 1965, the first case of proved FMH in the internal carotid arteries was reported.<sup>1</sup> Three patients with bilateral FMH of the extracranial internal carotid arteries have been studied at the University of California Medical Center. All 3 were females, and cerebral angiography was performed because of cerebrovascular

symptoms or because of asymptomatic bruits over the carotid arteries in the neck. The lesions in the extracranial internal carotid arteries were considered characteristic of FMH, but, to date, no operation has been performed, and the lesions have not been verified microscopically. One of the patients has proved FMH in the renal arteries.

The following case report outlines the pertinent features of one of these 3 patients.

**CASE II.** A 68 year old white woman was admitted to the University of California Medical Center in November, 1963 with a chief complaint of intermittent dizziness.

The results of the physical examination were unremarkable except for bilateral carotid bruits in the neck.

Aortic arch arteriograms and bilateral carotid arteriograms demonstrated unusual changes in

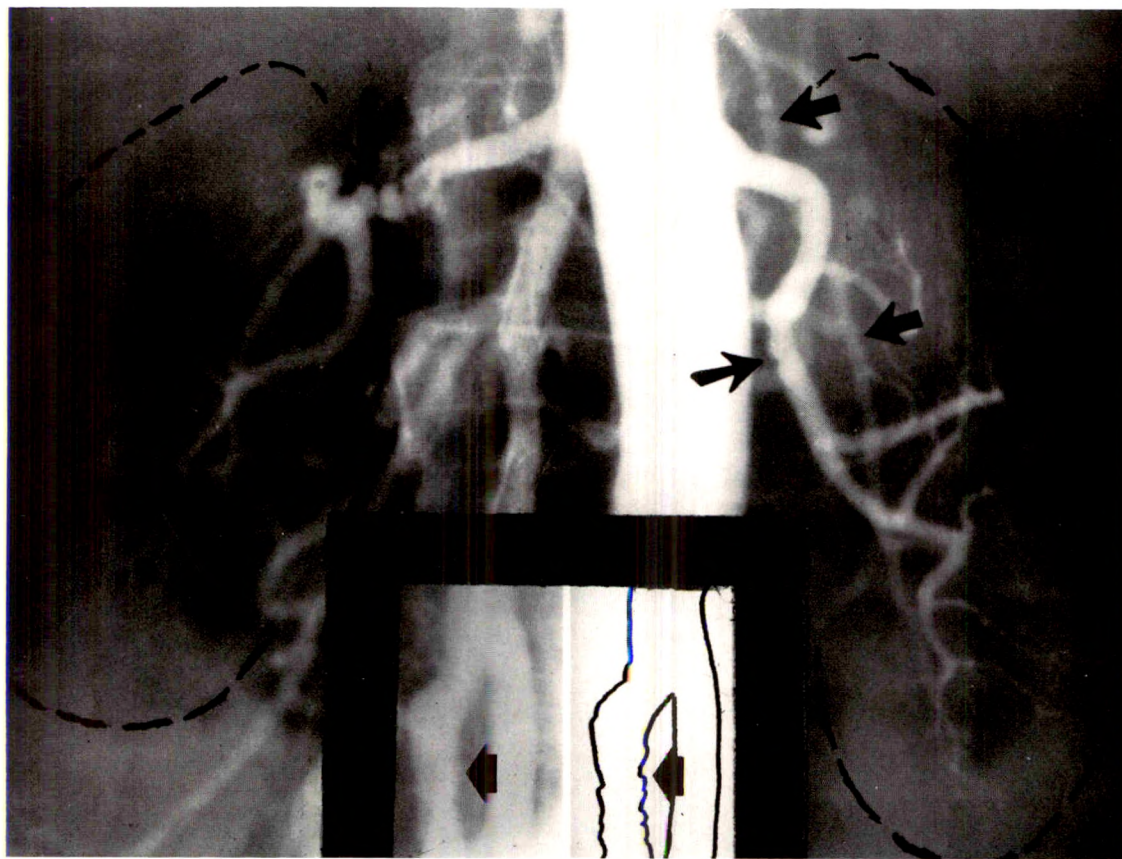
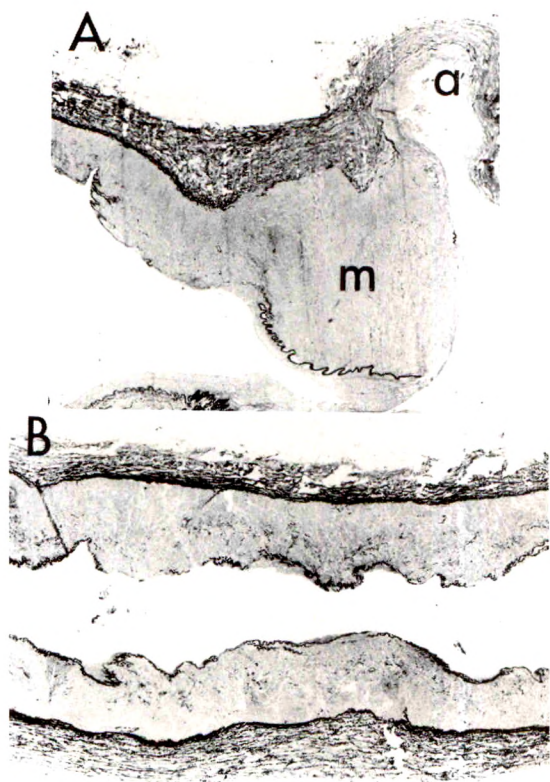


FIG. 2. Case 1. Proved fibromuscular hyperplasia (arrows) of the right renal, both left renal, and the right external iliac arteries (insert).





both extracranial internal carotid arteries, considered typical of FMH (Fig. 4, *A* and *B*).

The patient was discharged with the diagnosis of cerebrovascular insufficiency secondary to FMH of the internal carotid arteries. To date, no reconstructive procedures to the carotid vessels have been performed.

#### FMH AND INTRACRANIAL ANEURYSMS

The exact incidence of cerebral aneurysm is not known, but most authorities accept a figure of 2 per cent as the probable incidence in the general population.

Of the group of 70 patients studied at the University of California Medical Center with FMH of the renal arteries, 5 have one or more intracranial aneurysms. Of



FIG. 3. Case 1. Microscopic appearance of the fibromuscular hyperplasia in the (*A*) right renal and (*B*) right external iliac arteries. Fibromuscular hyperplasia was also present in the celiac and superior mesenteric arteries. a = aneurysm; m = hyperplastic media. Magnification: Renal artery ( $\times 26$ ); iliac artery ( $\times 15.5$ ).

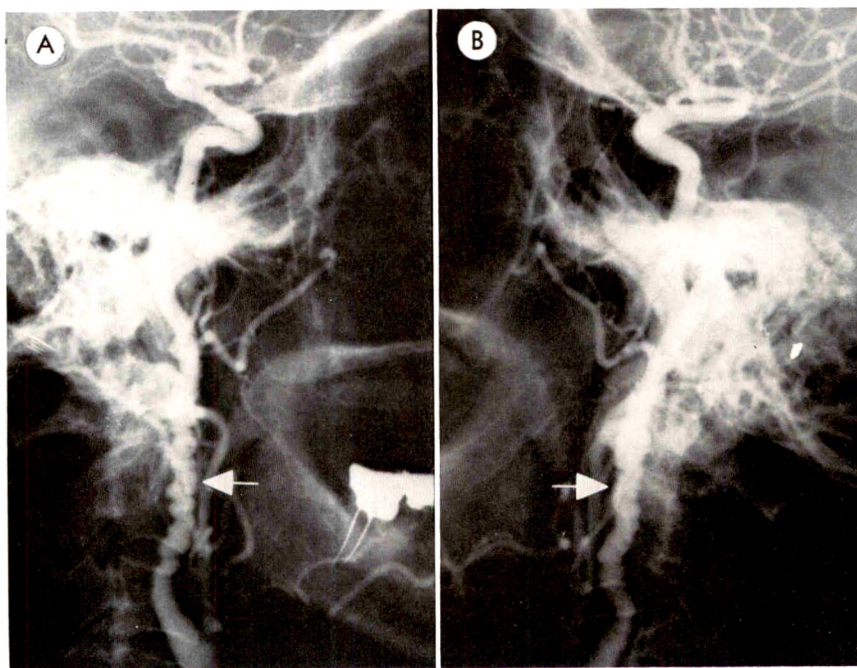


FIG. 4. Case II. (*A* and *B*) Roentgenographic appearance of both extracranial internal carotid arteries, considered diagnostic of fibromuscular hyperplasia (arrows). Compare with proved fibromuscular hyperplasia of the renal artery in Figure 1*A*. Note the selective symmetric involvement of the paired arteries.



these 5 patients, the FMH of the renal arteries was proved in 4 and was inoperable in the fifth.

A sixth patient with the roentgen appearance of FMH in both extracranial internal carotid arteries and an intracranial aneurysm has also been seen. To date, this patient's renal arteries have not been studied.

In the following case report the pertinent features in one of these 6 patients is outlined.

CASE III. FMH of the renal arteries had been proved in this 52 year old white woman. The same lesion had been diagnosed angiographically in branches of the mesenteric and celiac arteries. The patient was re-admitted to the University of California Medical Center in

May, 1965 for investigation of bilateral carotid artery bruits in the neck. These had caused no symptoms.

Aortic arch studies and subsequent carotid arteriograms demonstrated the appearance of FMH of both extracranial internal carotid arteries. An intracranial aneurysm of the right internal carotid artery was also present (Fig. 5, *A* and *B*).

No surgical procedure was performed and the patient was discharged asymptomatic to the care of her private physician.

Figure 6 demonstrates the findings in another patient whose arteriograms showed FMH in the right renal and external iliac arteries (Fig. 6, *C* and *D*) and multiple intracranial aneurysms (Fig. 6, *A* and *B*). The patient was a 58 year old woman with

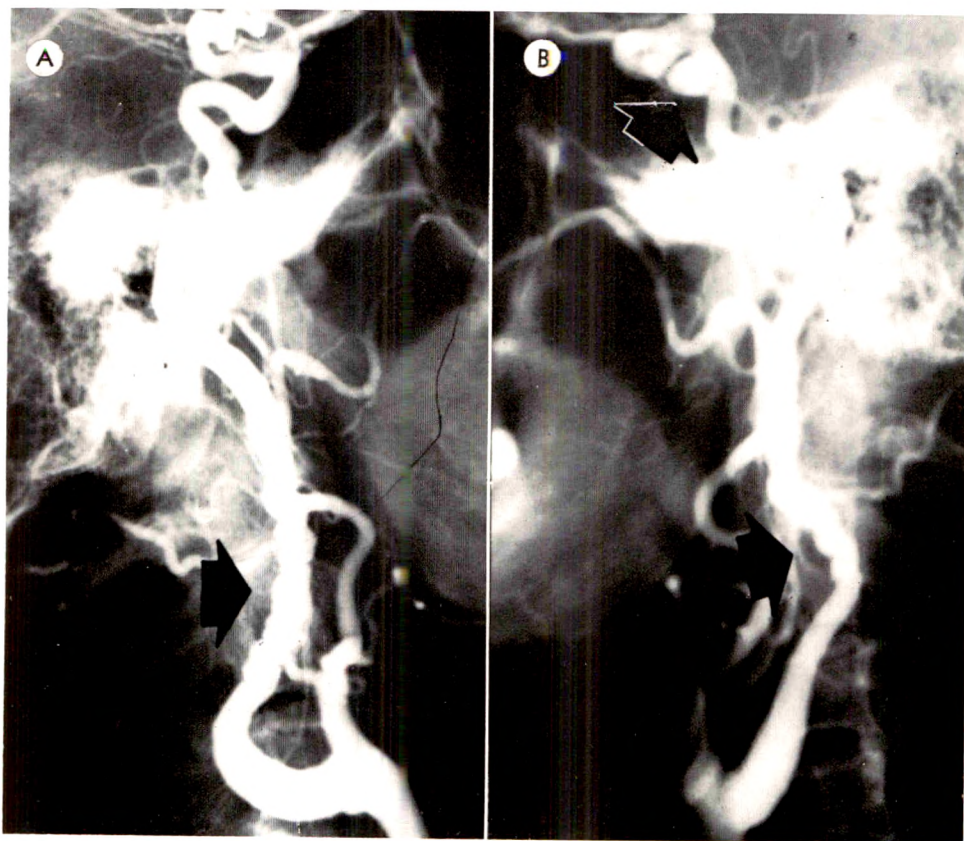


FIG. 5. Case III. (*A* and *B*) Angiographic changes considered typical of fibromuscular hyperplasia (lower arrows) of both extracranial internal carotid arteries. An intracranial aneurysm (upper arrow in *B*) of the right internal carotid artery is demonstrated. Fibromuscular hyperplasia of the renal arteries was proved in this patient and the roentgenograms showed a similar lesion in the celiac and superior mesenteric arteries. Note the selective involvement of both extracranial internal carotid arteries.



hypertension. To date, no operation has been performed and the roentgenologic diagnosis of FMH in the renal and external iliac arteries has not been proved.

#### DISCUSSION

In the last decade a resurgence of interest in renal ischemia as a cause of serious hypertension has highlighted the importance of FMH as a cause of renal artery stenosis and secondary high blood pressure. Only recently, however, has it been shown that the lesion is not limited to the renal arteries. Single cases of involvement of the celiac artery<sup>6</sup> and of the internal carotid artery<sup>1</sup> have been reported. Case 1 of this report represents an example of proved FMH lesions in multiple abdominal arteries, including the renal.

The coexistence of both symptomatic

and asymptomatic intracranial aneurysm in patients with FMH has not been investigated. The unexpectedly large number of intracranial aneurysms in the present series (8.5 per cent) in all likelihood is not coincidental. It probably reflects a relationship between the arterial dysplasia and intracranial aneurysms. Fewer than 15 per cent of the 70 patients here reported have had cerebral angiography. An effort is now being made to obtain elective carotid arteriograms in all patients with arterial FMH.

#### SUMMARY

Fibromuscular hyperplasia (FMH) is an arterial dysplasia of unknown etiology which can affect muscular arteries of small or moderate size in many parts of the body. It is most often discovered in the renal

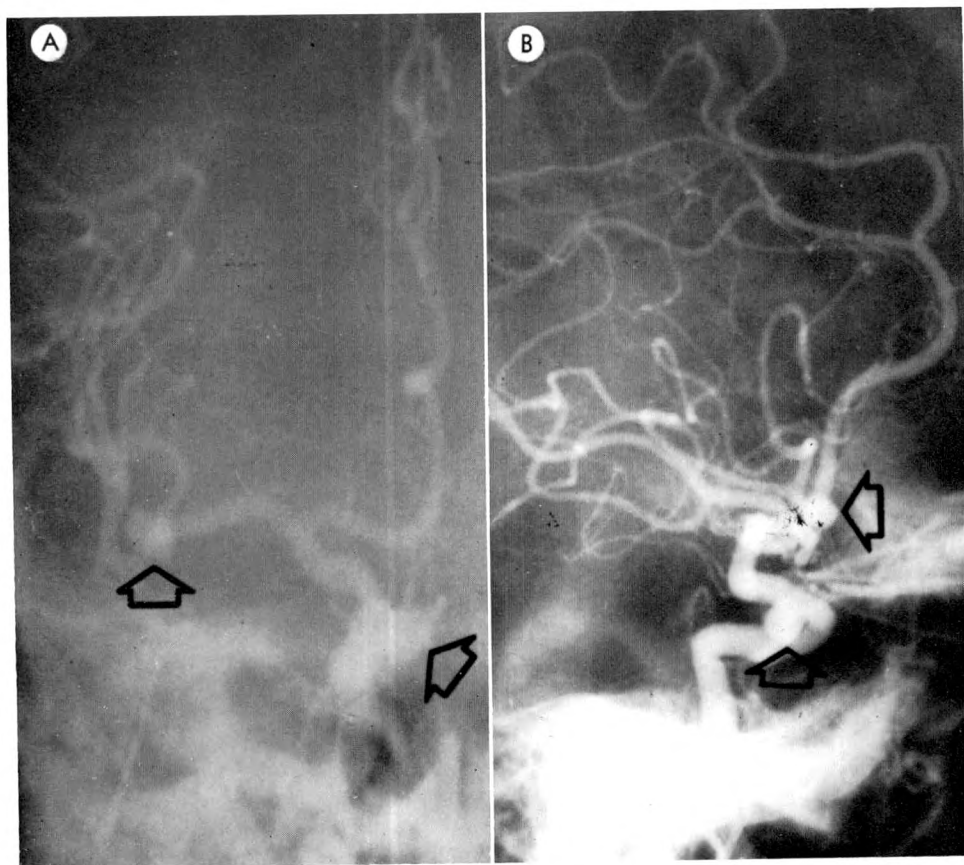


FIG. 6. (A and B) Right intracranial aneurysms (arrows) in a patient with the roentgenologic diagnosis of FMH of the right renal and both external iliac arteries. The patient had a third intracranial aneurysm on the left.

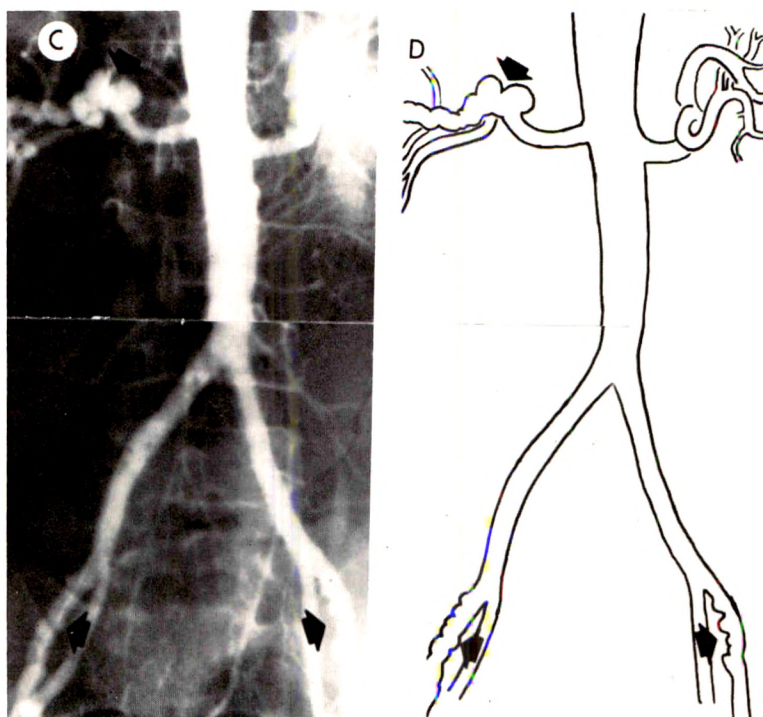


FIG. 6. (C and D) Arteriographic appearance of fibromuscular hyperplasia (arrows) of the right renal artery. Compare with proved fibromuscular hyperplasia in Figure 1B. Note selective symmetric involvement of external iliac arteries with normal aorta and common iliac and internal iliac arteries.

arteries of young, hypertensive adult females. Bilateral involvement of paired arteries appears to be common.

Case reports of 3 patients are presented to illustrate the occurrence of the lesion in extrarenal arteries. The incidence of both symptomatic and asymptomatic intracranial aneurysms in a group of patients with arterial FMH was unusually high.

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## BLOOD PRESSURE CHANGES INDUCED BY SUBCLAVIAN INJECTION OF METHYL- GLUCAMINE DIATRIZOATE (RENOGRAFIN)\*

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**C**ORRECT diagnosis of disease of the larger extracranial arteries as a cause of cerebral vascular symptoms is important since such a condition may be remedied by surgery with prevention of cerebral damage. To diagnose this condition, arteriographic visualization of the large arteries in the upper thorax and neck is widely employed. Demonstration of the great vessels such as the subclavian and innominate arteries, together with the origin of the carotid or vertebral arteries, is usually achieved by mechanical injection of large boluses of contrast material at high pressure. The occasional occurrence of reflex hypotension is a calculated risk.

Pharmacologic properties of contrast media including certain general cardiovascular responses have been reported by many authors but it might be supposed that rapid injection of large amounts of contrast material into the cephalic circulation could induce reflex responses of a neurogenic type. This latter possibility has not been adequately investigated.

The present study was designed to examine any changes in blood pressure following injection of methylglucamine diatrizoate (Renografin) into the subclavian artery and to analyze the mechanisms of any such changes.

### MATERIALS AND METHODS

Twenty adult mongrel dogs of either sex weighing 8–12 kg. were used. Each animal was anesthetized with intravenous pento-

barbital sodium in doses of 35 mg./kg., which was supplemented when required. Atropine sulfate was *not* given. An endotracheal tube was inserted and the femoral vein was cannulated for administration of drugs and fluids.

Both carotid arteries, the right subclavian, internal thoracic, omocervical and femoral arteries were exposed. A PE 205 polyethylene catheter, 60 cm. long, was introduced into the subclaviann artery via the omocervical artery for injection of the contrast medium or saline, the tip of the catheter being placed at the origin of the right vertebral artery.

Blood pressure was recorded by Statham pressure transducers\* connected to catheters threaded into: (1) the abdominal aorta via the femoral artery, (2) the carotid bifurcation via the external carotid, and (3) the right subclavian artery via the internal thoracic artery. Records of blood pressures were made with a Grass Model V Polygraph.

When artificial respiration was necessary, a Harvard Variable Speed Respirator† was used. Immobilization was obtained by intravenous injection of 5–7 mg./kg. of gallamine triethiodide.‡ Injection of the contrast medium or saline was performed by a Cook Automatic Pressure Injector§ with a 10 cc. metal syringe set at gauge

\* Statham Instruments, Puerto Rico, Model P23DB.

† Harvard Variable Speed Respirator, Model 606, Harvard Apparatus Co., Inc., Dover, Massachusetts.

‡ Flaxedil, Davis & Geck, Danbury, Connecticut.

§ Cook Inc., 300 Swain Ave., Bloomington, Indiana.

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† Fellows of the Michigan Heart Association. Dr. Handa's present address is the Department of Neurosurgery, Kyoto University Medical School, Kyoto, Japan.

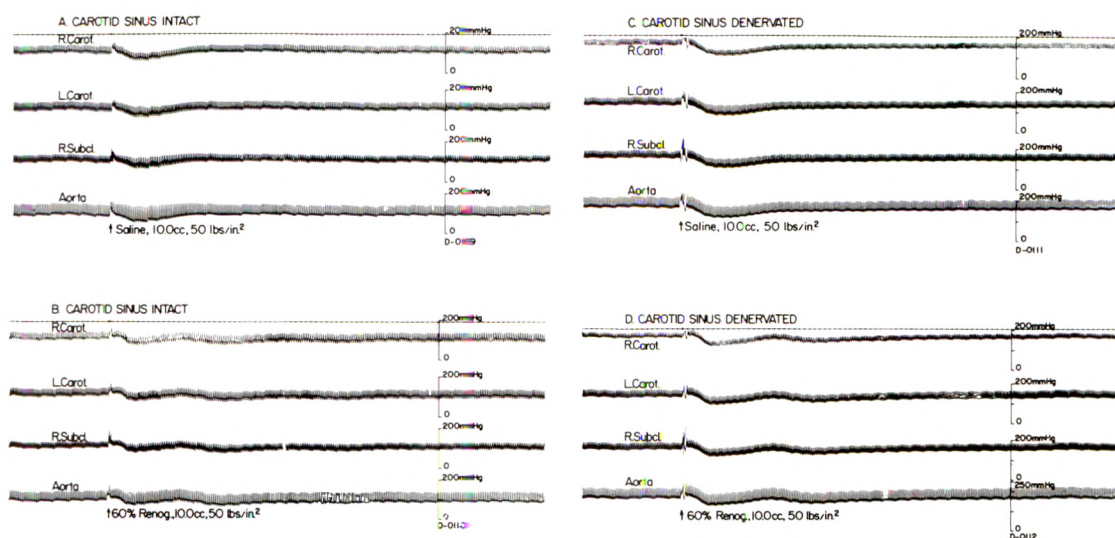


FIG. 1. Effect of injection of saline (*A* and *C*) and 60 per cent renografin (*B* and *D*) into the right subclavian artery on blood pressures in the right and left carotid, and right subclavian arteries and the aorta. Depression of blood pressure was observed after injection and was augmented after bilateral carotid sinus denervation. Time scale is in seconds.

pressures ranging from 5 to 50 lbs./in.<sup>2</sup>.

In several dogs the common carotid or femoral artery flow was measured continuously by means of a Microflo electromagnetic flowmeter.||

## RESULTS

### GENERAL PATTERN OF BLOOD PRESSURE RESPONSE

Immediately after the injection of 60 per cent methylglucamine diatrizoate (renografin) into the right subclavian, the blood pressures in the right subclavian, both carotid arteries and aorta all increased sharply but transiently.

Elevation of the blood pressure was greatest in the subclavian artery. The magnitude of increase in blood pressure within the carotid arteries and the aorta, however, was not the same. Carotid and aortic blood pressures often showed a short delay before the elevation (Fig. 1, *A-D*).

The blood pressure returned to the pre-injection levels in 2–5 seconds, followed by a period of hypotension lasting for 10 seconds to 2 minutes, after which the pressure regained pre-injection levels. The second

phase of hypotension was often associated with an increased pulse pressure and bradycardia. In some cases, the hypotensive phase was biphasic (Fig. 1, *B* and *D*).

During the first injections in 3 dogs, no depression of the blood pressure was observed but it remained elevated for 10–30 seconds before returning to the pre-injection levels. In the same 3 dogs, however, typical hypotensive responses were observed in later experiments. This monophasic response has also been described by Lindgren and Törnell<sup>6</sup> and Lin, Kricheff and Chase,<sup>5</sup> but its mechanism is not clear.

### INITIAL INCREASE IN BLOOD PRESSURE

The degree of blood pressure elevation correlated with both the injection pressure (gauge pressure) and the volume of renografin injected. Results with 1, 2, 3, 5 and 10 cc. of 60 per cent renografin at 50 lbs./in.<sup>2</sup> are shown in Figure 2*A*. Artificial respiration was not used in this series.

The magnitude of blood pressure elevation was proportional to the volume of contrast material injected within the range of 1–5 cc. The rise of blood pressure following injection of 10 cc. of 50 per cent renografin,

|| Medicon Division of Statham Instruments, Inc., Los Angeles, California. Model M-4001.



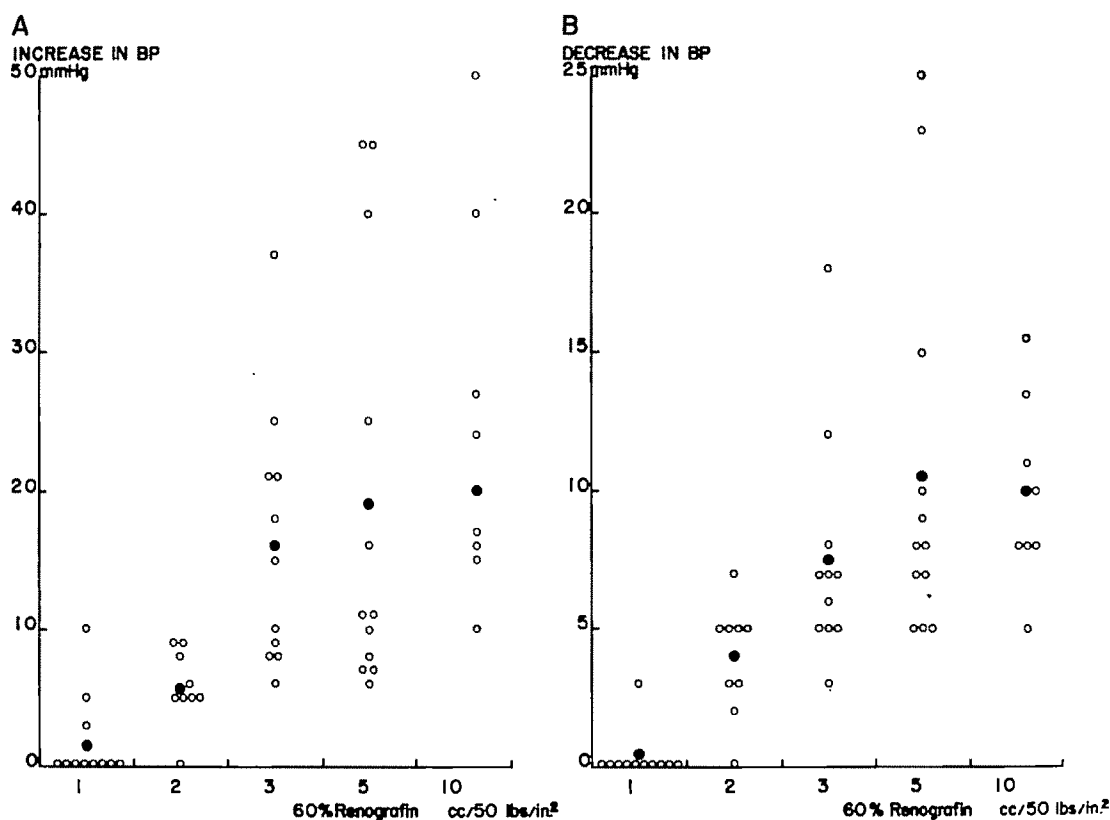


FIG. 2. Relationship between the magnitude of blood pressure elevation (*A*) and of secondary hypotension (*B*), and the volume of 60 per cent renografin injected.

○ = Mean of 2-3 experiments in each dog.

● = Mean of all experiments.

however, was much the same as the increase following 5 cc., although after injection of 10 cc. or more, the duration was longer.

When the injection pressure was increased from 5 lbs./in.<sup>2</sup> to 50 lbs./in.<sup>2</sup> with the volume constant, the increase of blood pressure was proportional to the gauge pressures.

Similar results were obtained with injection of saline. The magnitude of blood pressure elevation was approximately the same for either saline or 60 per cent renografin, provided the volume and the injection pressure were the same.

#### SECONDARY DEPRESSION OF BLOOD PRESSURE

Maximal decreases of blood pressure following injection of 1, 2, 3, 5 and 10 cc. of 60 per cent renografin at 50 lbs./in.<sup>2</sup> were plotted (Fig. 2*B*). No depression of blood

pressure was observed after injection of 1 cc. of the contrast material except once. When the volume was increased to 2, 3 and 5 cc., hypotension became progressively greater, but the decrease in blood pressure after injection of 10 cc. of the contrast material was much the same as following 5 cc. Hypotension tended to last longer, the larger the amount of contrast material used (Fig. 2*B*).

In 5 out of 15 dogs, the blood pressure decreased significantly following the injection of normal saline (Fig. 1*A*); in the remaining 10 no significant decrease of blood pressure was observed.

#### EFFECT OF CAROTID SINUS DENERVATION ON SECONDARY HYPOTENSION

In 8 dogs, the hypotension was compared before and after the denervation of the

carotid sinuses. Completeness of denervation was checked by noting the elevation of blood pressure level following sinus nerve section and also by temporarily occluding the common carotid artery and noting abolition of any blood pressure change. In this group respiration was maintained mechanically.

In 7 experiments hypotension was more marked after sinus denervation (Fig. 1, *A* and *C*; and 1, *B* and *D*). In 3, hypotension did not change significantly, the changes being only 1, 1, and 3 mm. Hg, respectively (Fig. 3*A*). Although the duration of hypotension was variable between animals, it tended to become more prolonged after sinus denervation.

#### EFFECT OF VAGOTOMY ON SECONDARY HYPOTENSION

In 5 dogs, hypotension was compared before and after bilateral vagotomy. Each

animal was tested both for 60 per cent renografin and normal saline. The decrease in the blood pressure became greater in only 1, became smaller in 6, and remained much the same in 3 (Fig. 3*B*).

When vagotomy was performed bilaterally in addition to previous carotid sinus denervation, hypotension became greater in 2 and lower in the remaining 3 (Fig. 4*B*). When compared with the response with both carotid sinus and vagus nerves intact, the degree of hypotension was always greater after sinus denervation plus vagotomy (Fig. 3*A*).

#### EFFECT OF TRANSECTION OF THE CERVICAL SPINAL CORD

In 5 artificially ventilated dogs, carotid sinus denervation and vagotomy were performed bilaterally and the spinal cord was transected at C1. Normal saline containing

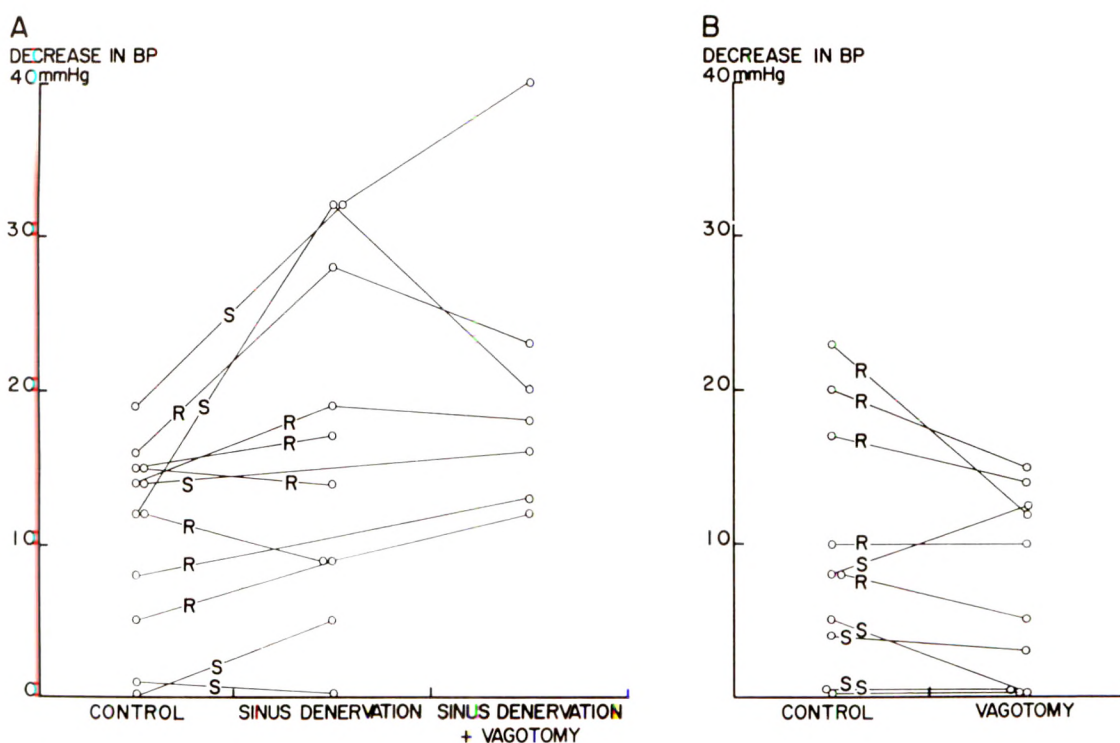


FIG. 3. Effect of carotid sinus denervation, and sinus denervation plus vagotomy (*A*), and vagotomy (*B*), on the magnitude of blood pressure depression.

○ = Mean of 2-3 experiments in each dog.

S = Injection of 5 cc. of normal saline at 30 lbs./in.<sup>2</sup>.

R = Injection of 5 cc. of 60 per cent renografin at 30 lbs./in.<sup>2</sup>.



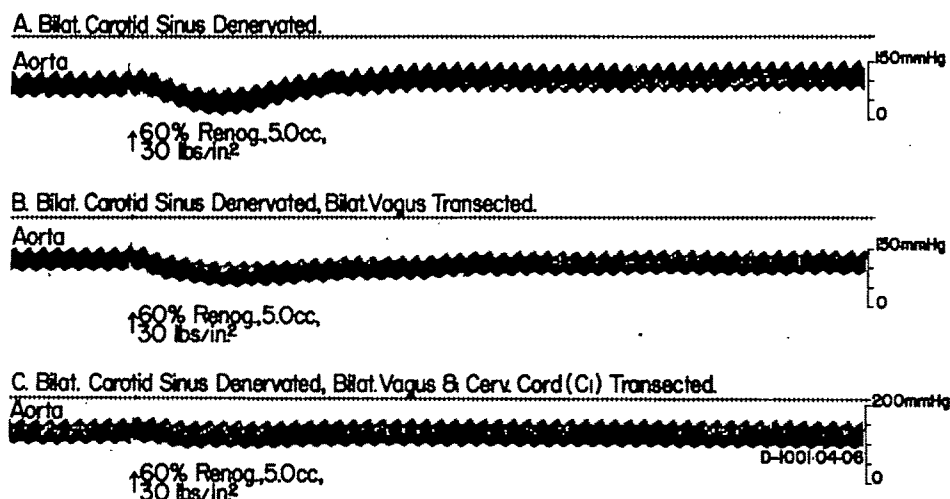


FIG. 4. Effect of injection of 60 per cent renografin on aortic blood pressure after bilateral carotid sinus denervation (A), sinus denervation plus vagotomy (B), and sinus denervation, vagotomy plus spinal cord section at C1 (C).

a small amount of epinephrine was administered by a slow intravenous drip to maintain blood pressure.

The hypotensive phase following the injection of renografin was abolished completely by spinal cord transection in 3 animals, and in 1 the magnitude of decrease in the blood pressure was reduced to a minimum (Fig. 4C). In the remaining 1, hypotensive responses of moderate degree were still observed after cord section.

#### CHANGES IN COMMON CAROTID AND FEMORAL ARTERY FLOW

Following the injection of renografin, the blood pressure decreased in all 6 dogs in which the blood flow was measured either in the common carotid or femoral artery. In 4 dogs the flow in both arteries increased moderately or remarkably during the hypotensive phase (Fig. 5C) and in 2 the arterial blood flow remained unchanged despite significant hypotension (Fig. 5B). In no instance was there any decrease in flow.

Following the injection of normal saline, a decrease in the blood pressure was not observed in 5 dogs, and the arterial flow also remained unchanged. In 1 dog, the blood pressure showed a decrease in a few experiments and remained unchanged in

the other. The increase in arterial flow was observed only in cases where the blood pressure decreased (Fig. 5A).

#### DISCUSSION

A transient elevation of the blood pressure immediately after injection of either renografin or saline appears to be attributable to hydraulic factors accompanying the forceful injection of a large bolus of fluid at high pressure, rather than to specific neurogenic factors or pharmacologically determined cardiovascular effects.

In the present study, injection was made into the subclavian artery at a high pressure, and it seemed probable that the injection affected the sinus nerve and/or the aortic arch depressor nerve mechanism.

However, the results of bilateral carotid sinus denervation indicated that the carotid sinus reflex is not a primary factor causing hypotension. This conclusion is in agreement with the results of Browne and Stern,<sup>1</sup> Lindgren and Törnell,<sup>6</sup> Robb and Steinberg,<sup>8</sup> and others. It by no means follows, however, that the carotid sinus reflex plays no role in the blood pressure response after angiography. When the responses were compared before and after sinus de-

nervation, hypotension was greater and more prolonged after denervation. This was attributed to the loss of the baroreceptive reflex of the carotid sinus.

Although the bradycardia can be ascribed to the vagal mechanism, the vagus nerves alone could not explain the hypotensive response.

It is well known that the injection of radiopaque media is followed by dilatation of the vascular beds in most of the organs, including the brain.<sup>3,9,10</sup> Thus, in reporting a fall of the blood pressure following angiography, Dotter and Steinberg,<sup>2</sup> Lin, Kricheff and Chase,<sup>5</sup> and Maluf and McCoy<sup>7</sup> all attributed the hypotension to peripheral vasodilatation. It has been stated that the vasodilatation is due to direct action of the contrast media on the smooth muscles of the vessels.

In the present study, a significant rise of

carotid and femoral artery flow after injection of renografin was confirmed. However, when the same amount of contrast material was injected into the aorta, the increase in peripheral blood flow was accompanied by hypotension of a much smaller degree. Furthermore, injection of normal saline into the subclavian artery also resulted in a remarkable hypotensive response with an increased peripheral blood flow (Fig. 5A), which again was not reproduced when saline was injected into the aorta.

In another series of experiments reported elsewhere,<sup>4</sup> the systemic blood pressure and cerebral blood flow were measured during intracarotid injection of renografin in Rhesus monkeys. The carotid sinuses were denervated and a PE 90 polyethylene catheter was introduced into the carotid bifurcation via the external carotid artery for

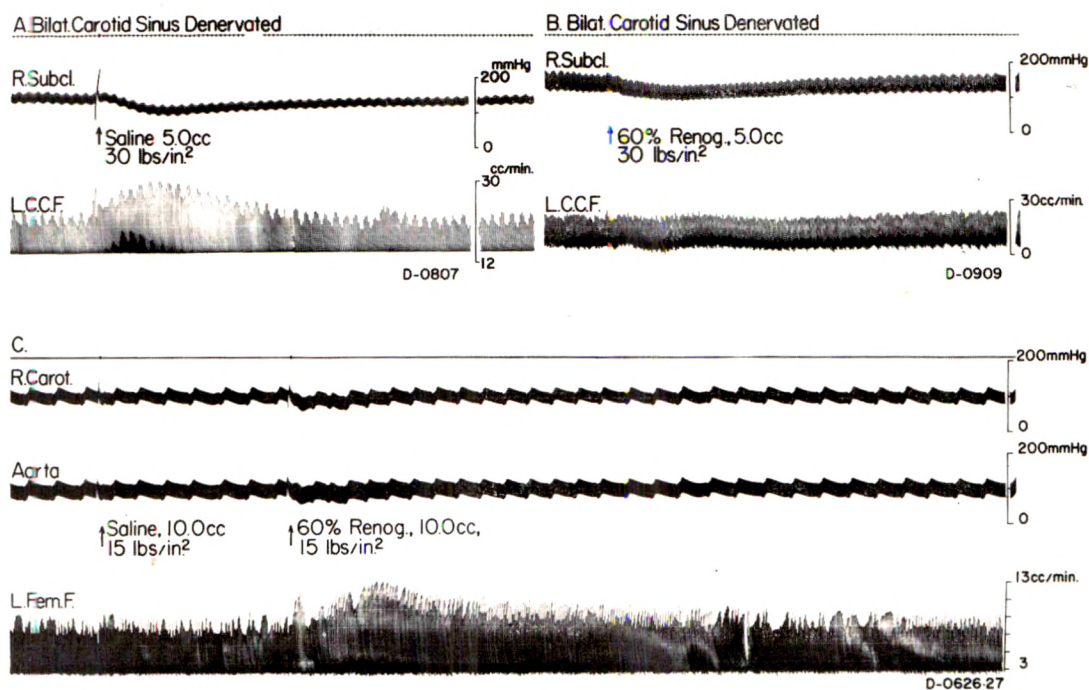


FIG. 5. Effect of intrasubclavian injection of saline and 60 per cent renografin on blood pressure and left carotid or femoral artery flow. In A, saline injection resulted in blood pressure depression and an increase in common carotid flow. In B, after injection of 60 per cent renografin, the blood pressure decreased significantly and the common carotid flow remained stable. In C, saline injection did not result in any blood pressure decrease and the femoral artery flow remained stable. Following 60 per cent renografin injection, the blood pressure showed a decrease associated with an increase in femoral artery flow. Time scale in seconds.



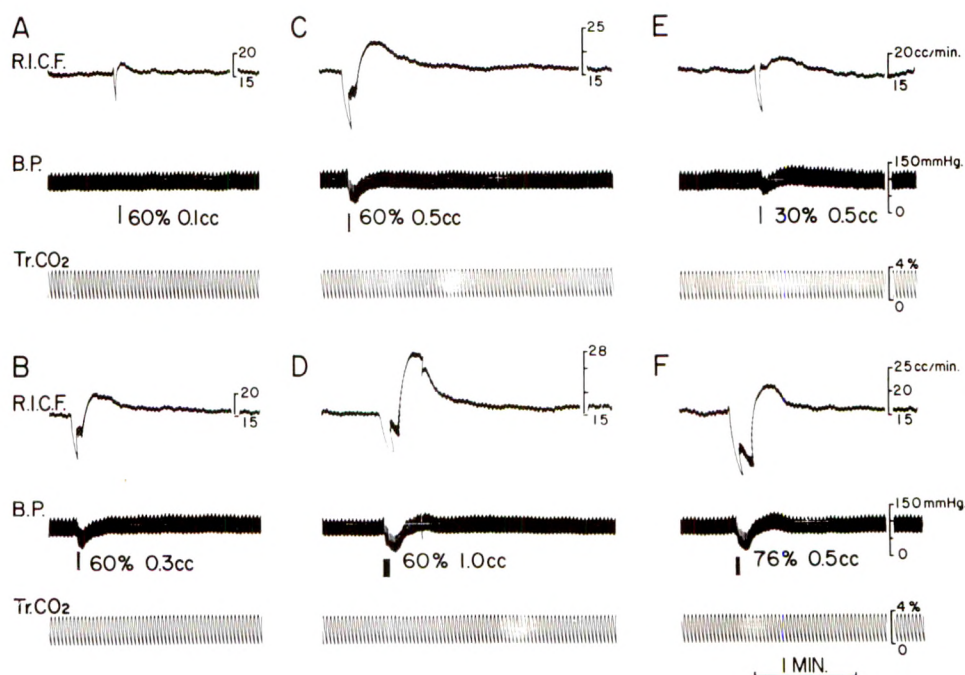


FIG. 6. (A-F) Effect of intracarotid injection of 60 per cent renografin on internal carotid blood flow and systemic blood pressure (aorta) in the Rhesus monkey.

injection. As shown in Figure 6, A-F, hypotension associated with bradycardia and an increase in cerebral blood flow was observed.

With this experimental set-up, backflow of the contrast material into the common carotid artery during retrograde injection is possible, as indicated by a temporary downward deflection of the flowmeter readings. (The flowmeter probe was placed on the common carotid artery with its external carotid branches ligated). When the small amount of contrast medium used was considered (for example 0.3 cc. in Fig. 6B), the severe hypotension with no latent period after injection could hardly be attributed to systemic redistribution of the medium with systemic vasodilatation due to direct effects of the contrast material on vessels. Moreover, intravenous injection of the same amount of contrast medium resulted in hypotension of lesser degree.

Under the conditions of retrograde carotid artery injection as well as injection into the subclavian artery near the origin of

the carotid and vertebral arteries or directly into the carotid artery, it therefore seemed reasonable to assume that the contrast medium or saline passed through the cerebral vascular beds and influenced cerebral vasomotor centers.

In an attempt to study the role of the central nervous system as a possible cause of the blood pressure fall, the spinal cord was transected at C1 in addition to carotid sinus denervation plus vagotomy. In this preparation, the hypotensive response following the injection of renografin was abolished or reduced to a minimum in 4 out of 5 dogs.

This conclusion conforms with the findings of Lindgren and Törnell<sup>6</sup> and supports the view that the fall in blood pressure after intracerebral injection of fluid materials is mediated by nervous mechanisms which were evoked by perfusion of the cerebral vascular bed. The present study does not provide information concerning regions of the brain responsible for the central hypotensive response.

## SUMMARY

1. Transient increase in blood pressure followed by a secondary hypotensive response and bradycardia was observed after rapid injection of contrast material or saline into the subclavian artery in the dog. Peripheral vasodilatation was noted.

2. The transient and prompt elevation of the blood pressure was due to local mechanical (hydraulic) factors following the rapid injection of a large bolus of fluid at high pressure.

3. The carotid sinus reflex does not play a primary role in the production of the secondary hypotension. Section of the vagus nerves abolished the bradycardia and diminished the fall of blood pressure.

4. Transection of the spinal cord at C1 abolished the fall of blood pressure. It was concluded that under the experimental conditions described, the hypotension following intra-arterial injection of a liquid bolus resulted from stimulation of central vasomotor centers by perfusion.

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## THE OSMOTIC EFFECTS OF UROGRAFIN 76 PER CENT AND ISOPAQUE 60 PER CENT IN ANGIOCARDIOGRAPHY\*

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MODERN contrast media used in angiocardiology have a remarkably low toxicity. This has led to a tendency to increase the doses in these examinations and also to increase the number of injections in one diagnostic session. However, the procedure is not without risks and even deaths are reported following rapid intravascular injection of contrast media in patients. Such injections have a marked osmotic effect as shown by Cotrim in 1954.<sup>2</sup> Later Iseri, Kaplan, Evans and Nickel,<sup>3</sup> Standen, Nogrady, Dunbar and Goldbloom,<sup>4</sup> and others had also demonstrated marked osmotic effects of injection of contrast media, resulting in acute expansion of the plasma volume. The purpose of this investigation was to study the plasma volume expanding effect of the 2 contrast media currently used in our routine angiocardiological examinations.

### MATERIAL AND METHODS

The material consists of 48 unselected patients who had angiocardiology performed as part of a routine preoperative evaluation for cardiac disease. The patients' ages varied from 14 to 75 years; 15 were women and 33 were men. In one-half of the cases isopaque 60 per cent\* (sodium metrizoate with calcium and magnesium metrizoate added) was used and in the other half urografin 76 per cent† (sodium—methylglucamine amidotrizoate). In 7 patients only one injection of contrast medium was made, in 14 two injections, in 13 three injections, in 11 patients four, and in 3 five injections. Most of the injections were made into the left side of the heart, *i.e.*,

into the left atrium, left ventricle or aorta. In only 7 cases, the injections were made into the right side of the heart.

Usually, there was an interval of 10 to 15 minutes between each injection of contrast medium. The injections were made through gray Ödman-Ledin catheters or in a few instances through NIH-type catheters. The pressure in the injector was adjusted so that similar amounts of iodine per time unit were injected with 2 contrast media. The speed of the injection was approximately 35 ml./sec. The highest dose of contrast medium used in any patient was 3.5 ml. per kg. body weight. This high dose was divided into 4 or 5 separate injections. No single dose of contrast medium was higher than 1.2 ml. per kg. body weight, and when repeated injections were used no single dose was higher than 0.8 ml. per kg. body weight.

In each patient hematocrit readings were obtained immediately before the injection of contrast medium and approximately 1 minute after each injection and 15 and 30 minutes after the last injection of contrast medium. In a few cases, values were obtained as late as 8 hours after the injection. The blood for the tests was drawn through a catheter in the ascending aorta, and an International microcapillary centrifuge and reader were used to determine the hematocrit.

### RESULTS

The results are summarized in Figures 1, A and B; and 2. It is obvious that injections of both urografin 76 per cent and isopaque 60 per cent produced a considerable decrease in hematocrit values.

With urografin (Fig. 1A), the mean de-

\* Isopaque 60%, Nyegaard & Co. AS, Oslo, Norway.

† Urografin 76%, Schering A. G., Berlin, Germany.

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crease was 18.5 per cent from the control value obtained immediately before the injection of contrast medium. Individual variations were from 4 to 33 per cent. With isopaque (Fig. 1B), the mean decrease was 15.6 per cent with individual variations from 7 to 32 per cent following one single injection of contrast medium. The decrease

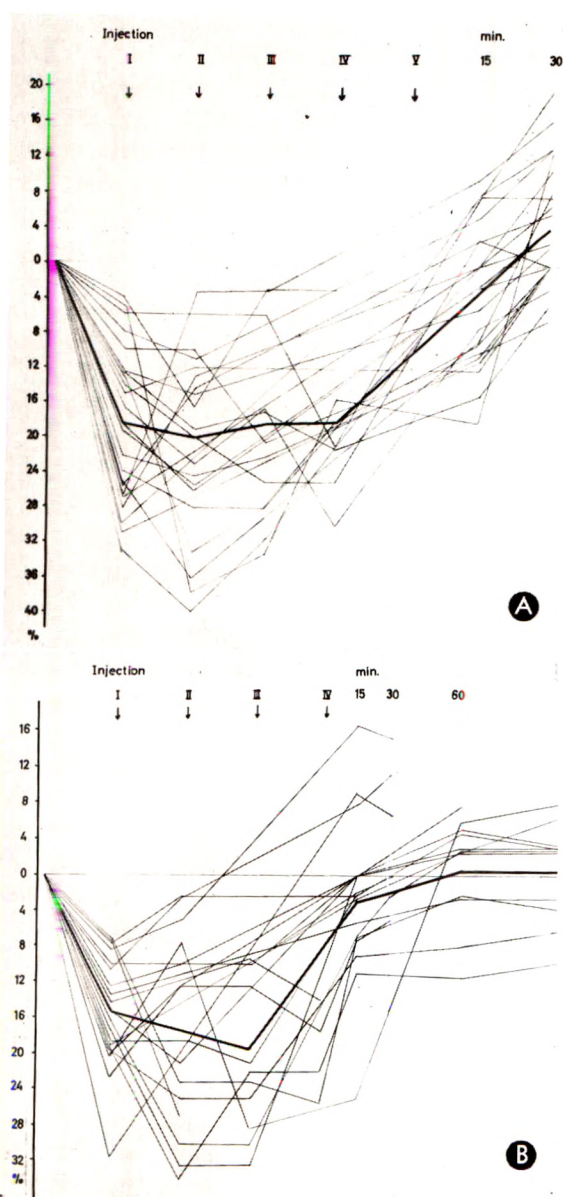


FIG. 1. Decrease in hematocrit (per cent of pre-injection values) following angiocardigraphy with (A) urografin 76 per cent and (B) isopaque 60 per cent.



FIG. 2. Decrease in hematocrit following angiocardigraphy (per cent of pre-injection values). Maximum decrease occurs approximately 60 seconds after injection.

following repeated injections was less pronounced with both contrast media. There was a fairly rapid restitution of the hematocrit values to the original ones. This was already seen 15 minutes after the last injection of contrast medium. The values were closer to normal and some of them above the original value. After 30 minutes, a tendency to "overshoot" was seen. In the cases studied for longer periods, moderate changes in hematocrit sometimes persisted for several hours after the injection of contrast medium.

In a special study, hematocrit readings were obtained as rapidly as possible following the injection of contrast medium and then with close intervals up to the first 4 minutes. It was found that the maximum changes in hematocrit occurred approximately 1 minute after the injection (Fig. 2).

No complications occurred in this series. The patients' subjective symptoms were a slight or moderate headache and a sensation of warmth following the injection of contrast medium. No vomiting or shock occurred. In 2 cases a slight allergic skin reaction was noted. Electrocardiographic changes of previously described type were noted in some cases, but they were all slight and transient. These slight subjective and objective side effects were no more frequent after repeated injections than after the first one, and they were equally frequent with both contrast media.

#### DISCUSSION

The decrease in hematocrit is mainly explained by an acute expansion of the



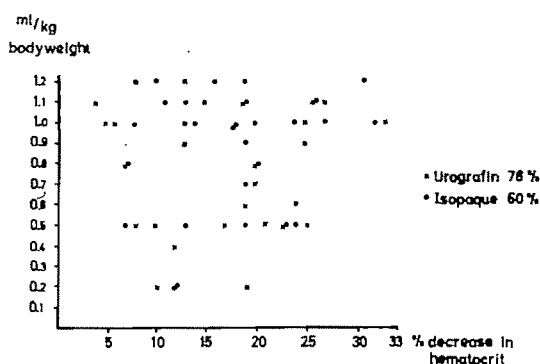


FIG. 3. Relation between dose of contrast material and decrease in hematocrit.

plasma volume caused by the injection of the hyperosmotic contrast medium into the vascular system of the patients. Iseri *et al.*<sup>3</sup> and Standen *et al.*<sup>4</sup> found good correlation between the increase in plasma volume and the decrease in hematocrit in similar studies. The changes in hematocrit found in this material were more marked than those previously reported. This may be explained by differences in size of material, injection techniques, techniques for obtaining samples for hematocrit determinations and dose of contrast medium. Standen *et al.*<sup>4</sup> studied 8 children following slow intravenous injection of average doses of 4.7 ml. per kg. body weight and the samples for hematocrit determinations were drawn from a peripheral vein. Iseri *et al.*<sup>3</sup> studied 8 patients after aortic injections of smaller amounts of contrast medium than those in the present study. However, in principle, the changes in hematocrit in this material and in the previous reports are similar. Following a rapid intravascular injection of contrast medium, there is a decrease in hematocrit, reaching the maximum approximately 60 seconds after the injection and then slowly returning to normal values within 15 to 30 minutes.

There were great individual variations in the decrease of the hematocrit following injection of similar doses of contrast medium in different patients. There is almost a complete absence of correlation between dose and the decrease in hematocrit. A

dose as small as 0.2 ml. per kg. body weight resulted in an 8 to 18 per cent decrease in hematocrit and a dose 5 times as high gave a decrease varying from 5 to 23 per cent (Fig. 3). Also, in the same patient there were variations in the decrease of hematocrit from time to time. In 1 patient identical injections of isopaque 60 per cent. were made within an interval of 1 week. During the first injection, the decrease in hematocrit was 25 per cent and during the second only 10 per cent. When the 10 patients with a decrease in hematocrit of 25 per cent or more were compared with the 9 patients with a decrease in hematocrit of less than 10 per cent, it was found that 8 patients in the first group but none in the second had a reduced physical working capacity. These 8 patients were also clinically assessed as in a poorer functional state than the 9 in the second group. Two of the 8 patients had slight pretibial edema on admission to the hospital and all 8 lost 2 kg. or more in weight during their first week in the hospital. This indicates the presence of an increased amount of free extracellular fluid in these patients and this could explain their marked hypervolemic response to injection of hyperosmotic contrast medium. There was no obvious correlation between decrease in hematocrit and age, sex or other clinical or laboratory data. Practically all of the pre-injection hematocrit values were within the normal range. Only 2 patients with tetralogy of Fallot had hematocrits above 60 and 1 woman had a hematocrit of only 28. The decrease in hematocrit in these 3 cases was not different from that in the others.

The effect of repeated injections was not as cumulative as expected (Fig. 1, A and B; and 2). This may be explained by the interval of 10 to 15 minutes usually occurring between the injections. In this interval, a normalization of the changes had time to occur. It is also possible that the sources of free extracellular fluid became exhausted by the first injection and, if this is true, the response to repeated injections would be less than to the first one.

It is obvious that very marked changes in the blood volume may occur following a single injection of contrast medium. These changes in blood volume are sufficient to explain the changes in the cardiac pressures and in cardiac output noted after angiocardiology (Brown, Rahimtoola, Davis and Swan,<sup>1</sup> and others). The sudden expansion of plasma volume immediately after the injection of contrast medium is sometimes considerable and could cause overdistention of the heart with precipitation of acute cardiac failure or initiation of arrhythmias. This mechanism could well explain some of the cases of death following angiocardiology.

It was remarkable that the patients withstood the sometimes very marked acute changes in plasma volume without obvious signs of distress and that no serious side effects occurred. However, one must be aware of the risk of serious complications following even small doses of contrast medium. Since the osmotic effects are unpredictable, close observation of the patient following each injection of contrast medium is necessary. This is particularly important in patients in a poor functional state with a tendency to edema, where the osmotic effects can be expected to be more marked than in other patients. Whenever there are clinical signs of acute hypervolemia or whenever intracardiac pressures are increased to unusually high level following angiocardiology, repeated injections of contrast medium should be avoided. If repeated injections are contemplated, an interval of at least 15 minutes should be allowed between injections. When serious complications occur following angiocardiology, it is important to know that such complications may be caused by acute hypervolemia. The treatment of such conditions must then, among other things, be directed against the hypervolemia.

It would seem possible that the headache experienced by some of the patients following angiocardiology may be explained by the osmotic effects of the contrast medium, causing dehydration of the

brain resulting in a decrease in intracranial pressure. In this material some of the patients with a moderate headache following angiocardiology had a marked decrease in hematocrit. However, the evaluation of a subjective symptom like headache is very difficult and the correlation between the osmotic effects of the contrast medium and the headache is uncertain.

The statistically significant difference between the decrease in hematocrit following injections of urografin 76 per cent and isopaque 60 per cent is interesting. This difference is explained by different osmotic activity of the 2 contrast media. There is a difference in size of the molecules and probably also a difference in degree of dissociation of the contrast medium in the blood. This difference in osmotic activity may, in part, explain the lower toxicity of isopaque 60 per cent compared to that of urografin 76 per cent found in animal experiments, particularly in small animals. However, in this clinical material the slight difference in osmotic activity had no observable effect.

It is obvious that the interpretation of angiograms obtained during repeated injections of contrast medium must be critical. If an interval of less than 30 minutes is allowed to pass between repeated injections of contrast medium, the hemodynamic conditions during the second injection may be different from the basic ones. This may particularly influence the evaluation of valvular function and the determination of the volumes of the cardiac chambers. It is also important to know that even a small test dose given to confirm the position of a catheter, under unfavorable conditions, may cause a considerable increase in the circulating blood volume and, consequently, change the hemodynamic status of the patient.

#### SUMMARY

In 48 patients, an average 17 per cent decrease of hematocrit values, indicating a considerable increase in circulating blood volume, was found after angiocardiology. The individual response varied from 4 to 33



per cent and was rather unpredictable, but the most severe changes were generally found in patients with a decreased physical working capacity and with a tendency to develop edema. The changes reached their maximum approximately 1 minute after the injection of contrast medium, and after 15 to 30 minutes there was generally a normalization of the hematocrit. The acute hypervolemia may explain some of the complications observed after angiocardiology and may also account for the difficulties encountered in the interpretation of angiograms obtained after repeated injections of contrast medium. Urografin 76 per cent gave an average decrease in hematocrit of 18.5 per cent as compared to a 15.4 per cent decrease with isopaque 60 per cent. The slightly higher osmotic activity of urografin 76 per cent made no observable difference in this clinical material.

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## THE EFFECTS OF ANGIOTENSIN ON RENAL VASCULARITY IN DOGS\*

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ANGIOTENSIN II (hypertensin II) is an octapeptide, presumably resulting from renal secretion of an enzyme (renin). Renin apparently arises from the juxtaglomerular apparatus of the kidneys in response to low renal blood flow and is related to the need of the kidney for additional filtration pressure. Renin acts on a plasma alpha-2-globulin (hypertensinogen) to produce a decapeptide (angiotensin I) which shows no constrictive activity on isolated blood vessels. Another enzyme in the blood stream (an angiotensin "activator") splits off the two terminal amino acids of the decapeptide to produce the active compound, angiotensin II. The terms angiotensin and hypertensin are used interchangeably. After elucidation of its chemical structure, angiotensin has been synthesized. The activity of synthetic angiotensin II is exactly the same as that in the natural form.

Synthetic hypertensin has been used clinically for the treatment of shock with variable reported results. del Greco and Johnson<sup>1</sup> reported that angiotensin is the most effective pressor agent available, with an activity 200 times that of metaraminol in respect to the pressor effect. In 3 of their 21 patients, there were data concerning urinary excretory function; the urine output was not reduced either during or following the administration of angiotensin II.

Nassif *et al.*<sup>10</sup> also reported favorably on the use of angiotensin II in the treatment of hypotensive states. They suggested that angiotensin probably increased and certainly did not interfere with urine flow.

Udhoji and Weil<sup>12</sup> questioned the usefulness of angiotensin for the treatment of

shock. They reported that the renal effects of angiotensin tend to accentuate the circulatory defect that occurs during shock—*i.e.*, reduction in renal blood flow, decreased glomerular filtration rate, increased filtration fraction and lowered urine volume. They concluded that the deleterious effects of angiotensin on renal function are more pronounced than those of the sympathomimetic amines.

Corcoran and Page<sup>2</sup> infused angiotensin into conscious, trained dogs with explanted kidneys. Their demonstration of increased arterial pressure, decreased renal blood flow and increased extraction of inulin from the blood suggested that the drug had produced vasoconstriction of the efferent glomerular arterioles.

Similarly, Finnerty<sup>6</sup> demonstrated a vasoconstrictive effect of angiotensin on post-glomerular segments. In subjects without cardiovascular disease, angiotensin infusion resulted in marked decrease in renal blood flow, moderate decrease in filtration rate, slight decrease in urinary volume and increase in filtration fraction.

Katz *et al.*<sup>8</sup> reported that subcutaneous administration of angiotensin to rats for a 1 to 2 week period produces histologic changes in the interlobar arteries and the afferent glomerular arterioles.

Byrom<sup>1</sup> reported that single intravenous injections of very large doses of angiotensin cause acute medial necrosis of large renal arteries in the rat.

Angiotensin apparently has a direct stimulant action on the smooth muscle of the vascular system. It acts on the vascular tree at a different site and through a different set of chemical receptors than do the catechol amines such as epinephrine and

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norepinephrine; its precise site and mode of action are unknown at present. In normotensive subjects it allegedly causes a decrease in glomerular filtration and urine output, whereas in patients in shock an increase in urine volume may occur.

Neri *et al.*<sup>11</sup> studied the effects of small doses of hypertensin II on the renal circulation in dogs, using 0.5 to 1.0  $\mu$ g. per kg. intravenously in single dose or 4 to 6  $\mu$ g. per minute in intravenous drip infusion. They performed aortography with an organic iodide contrast medium (urografin). They reported a constriction of the smaller renal arteries and glomerular capillaries with a marked delay of the nephrographic phase.

In previous studies,<sup>5,9</sup> renal angiography was used to investigate the effects of epinephrine, levarterenol and metaraminol on renal vascularity in the dog. In small doses epinephrine and levarterenol caused vasoconstriction of peripheral renal vessels with dilatation of the main renal artery and of the interlobar arteries, prolongation of the arterial phase with late filling of the veins, and diminution of the intensity of the vascular nephrogram. On the other hand, as far as could be judged from the angiogram, small intravenous doses of metaraminol (0.05 to 0.1 mg. per kg.) caused no decrease in renal blood flow. In large doses, all 3 of the tested sympathomimetic amines produced similar changes, suggesting increased renal vascular resistance with a major effect on the main renal arteries. There was sudden and marked constriction of the main renal artery at or near its bifurcation, attenuation of the interlobar arteries, decrease in intensity of the nephrogram, poor opacification of the intrarenal and main renal veins and evidence of slowed circulation.

In the studies now reported, angiotensin was investigated in respect to its effects on renal vascularity as compared to the actions of the sympathomimetic amines. In order to compare effects produced by angiotensin, levarterenol and metaraminol, it should be noted that the relative potency of these three drugs has been reported in the

literature in respect to their pressor response,<sup>3,6,7,10,12</sup> angiotensin being the most potent of the 3. As judged by weight, 1 unit of angiotensin produces a pressor effect equivalent to that produced by 1 to 17 units of levarterenol, and 30 to 100 units of metaraminol. The actual relationship of these drugs apparently varies with the hemodynamic state of the tested subjects. Thus, Finnerty<sup>6</sup> reported that in normotensive and hypertensive patients angiotensin is about 10 times as potent as levarterenol in changing arterial pressure and heart rate, whereas with patients in shock angiotensin is only 2 to 3 times more potent than levarterenol.

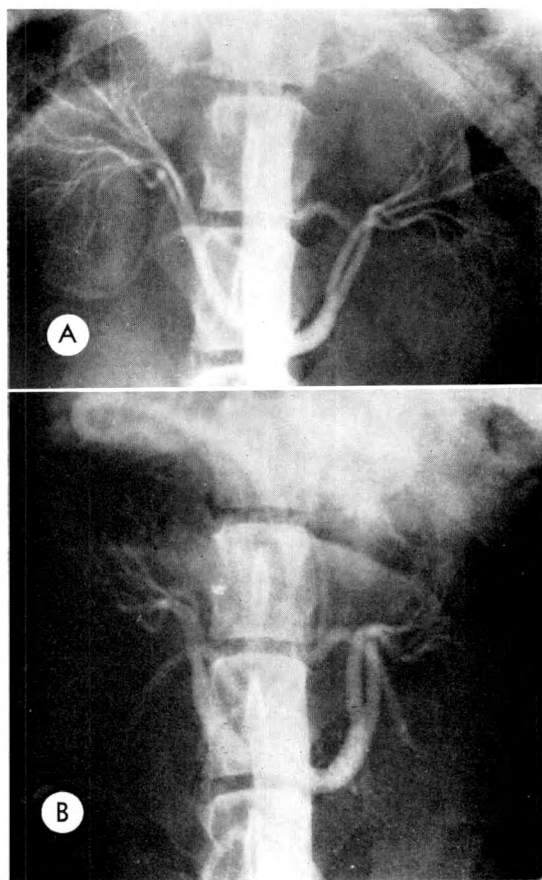


FIG. 1. (A) Control aortogram  $\frac{1}{2}$  second after the injection of thorotrast ( $\frac{1}{2}$  second film). (B) Thorotrast aortogram,  $1\frac{1}{2}$  second film, after intravenous administration of angiotensin, 5  $\mu$ g. per kg., shows dilatation of the aorta, the lumbar vessels, the main renal arteries as well as the interlobar arteries.

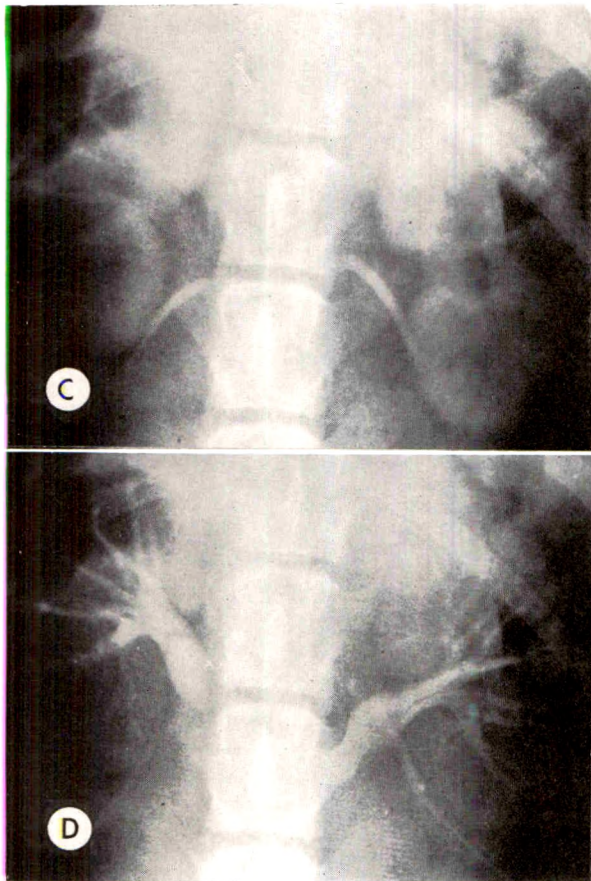
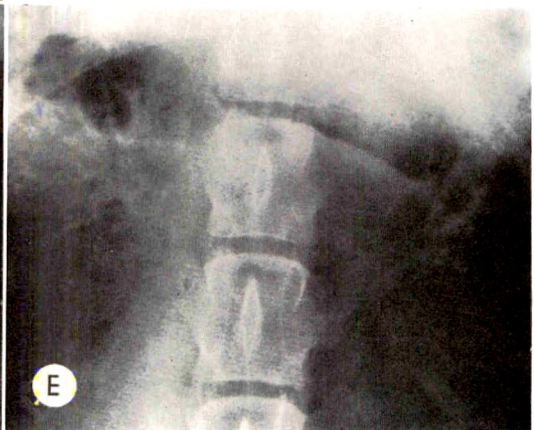


FIG. 1. (C) Control aortogram, 2 second film, shows good nephrogram bilaterally. (D) Control aortogram,  $3\frac{1}{2}$  second film, shows good venous opacification of both kidneys. (E) Post-angiotensin aortogram,  $3\frac{1}{2}$  second film (2 seconds after B), shows no nephrogram and, as yet, only poor venous opacification.



#### TECHNIQUE

As described previously,<sup>5</sup> total or selective renal angiography was done on dogs, using thorotrast as the contrast medium. Reproducibility of the injection factors was assured in respect to speed of delivery of the bolus of the opaque medium, amount of contrast material delivered and initiation of the roentgenographic exposures at the same time in the course of the injection. Intra-aortic blood pressure was continuously monitored during an experiment. Single dose intravenous administration of the tested drug was employed with the amount of angiotensin made up to 5 cc. with saline in all injections. Angiography was performed at the height of the blood pressure response, 30 to 60 seconds after completion of the intravenous injection. Hypertensin-CIBA (angiotensin amide) was used; this synthetic material has been shown to be

identical pharmacologically and physiologically to naturally occurring angiotensin II.

#### RESULTS

In dosage of 0.5 to 45  $\mu$ g. per kg., angiotensin produced similar changes, consisting of dilatation of the main renal and interlobar arteries, decrease in the intensity of the vascular nephrogram, prolongation of the phases of circulation with late and relatively poor opacification of the intrarenal and main veins. The prolonged arterial phase, the less intense nephrogram and the appearance of the venous segments all indicate diminished renal blood flow (Fig. 1, A-E). In no experiment was there constriction of the main renal artery or its major branches, as had been seen with the high doses of epinephrine, levarterenol and metaraminol. Regardless of dose, the vasoconstrictive action of angiotensin appears



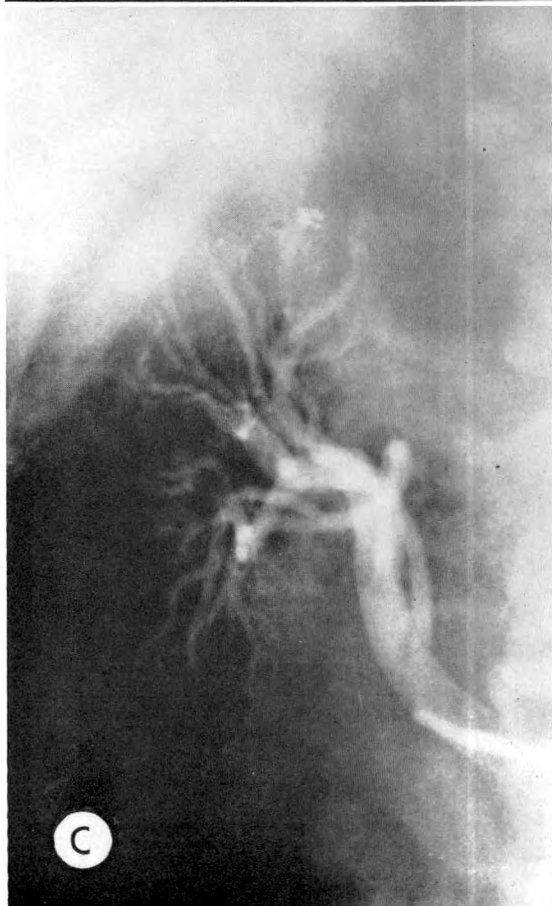
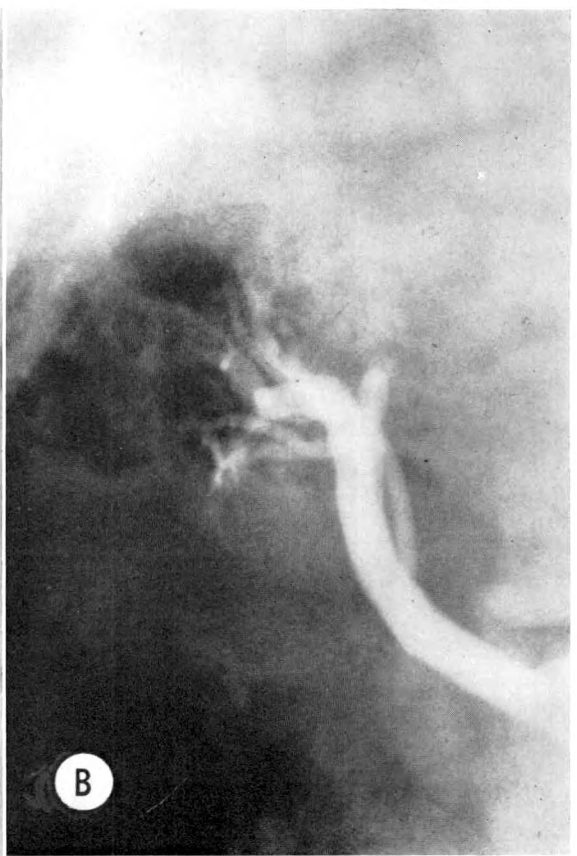
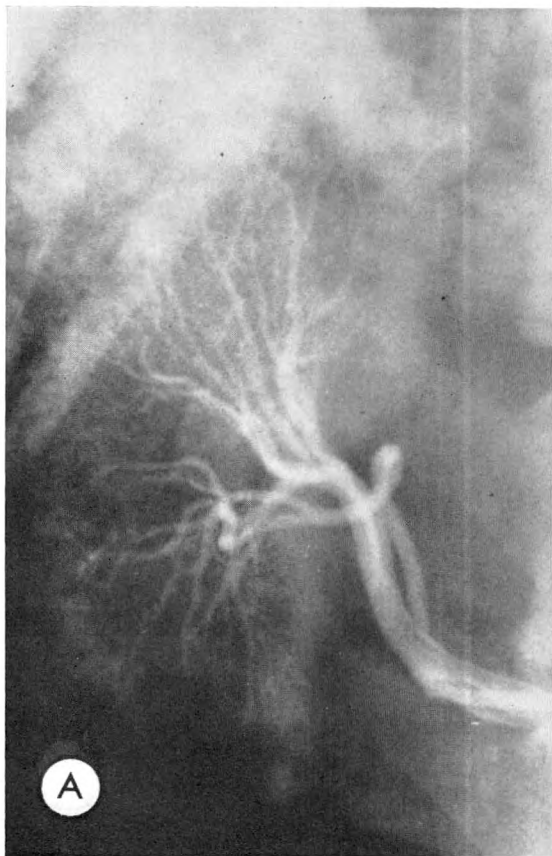


FIG. 2. (A) Control selective angiogram,  $1\frac{1}{2}$  seconds after the injection of thorotrast ( $1\frac{1}{2}$  second film). (B) Thorotrast angiogram,  $1\frac{1}{2}$  second film, after intravenous administration of angiotensin, 45  $\mu$ g. per kg., shows dilatation of the main renal artery with slower filling of dilated interlobar arteries. (C) Post-angiotensin angiogram, 3 second film ( $1\frac{1}{2}$  seconds after B), shows dilatation of the interlobar arteries. There is absence of the cortical nephrogram as seen in the  $1\frac{1}{2}$  second film of the control angiogram (A).

to be on vessels peripheral to the interlobar arteries (Fig. 2, *A*, *B* and *C*). In our normal dogs, the potency of angiotensin was similar to that of levarterenol in respect to increasing the mean blood pressure. Angiotensin in dose of 5  $\mu$ g. per kg. raised the mean blood pressure about 105 mm. Hg; levarterenol in dose of 5  $\mu$ g. per kg. caused a rise of about 85 mm. Hg. Yet, although angiotensin and levarterenol appear similar (on a weight-dose basis) in their effects on systemic blood pressure in the normal dog, levarterenol appears more potent in relation to effects on renal vascularity. Angiotensin in dose up to at least 45  $\mu$ g. per kg. produced changes seen with much smaller doses of levarterenol (e.g., 1  $\mu$ g. per kg.). Levarterenol in dose of 45  $\mu$ g. per kg. effected changes on the major branches of the renal artery; such dramatic changes were not seen with similar doses of angiotensin.

#### MECHANISM OF ACTION

Previous investigations<sup>5,9</sup> indicated that epinephrine, levarterenol and metaraminol have a direct action on the renal vessels and do not produce their effects merely by way of increased blood pressure. Similar studies were done with angiotensin. Bilateral selective renal angiography was done with a separate catheter introduced into each femoral artery, the thorotrast being injected simultaneously into both renal arteries through a Y-connector from the automatic injector to both catheters. After the control series, repeat bilateral angiography was carried out immediately after injecting angiotensin into the right renal artery, 45  $\mu$ g. per kg. The right side showed marked slowing of the progress of the opaque material so that at 4½ seconds there was still slight irregular filling of some of the peripheral renal arteries with no evidence of

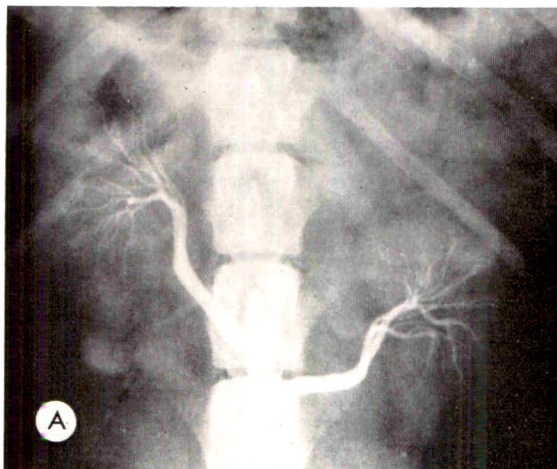
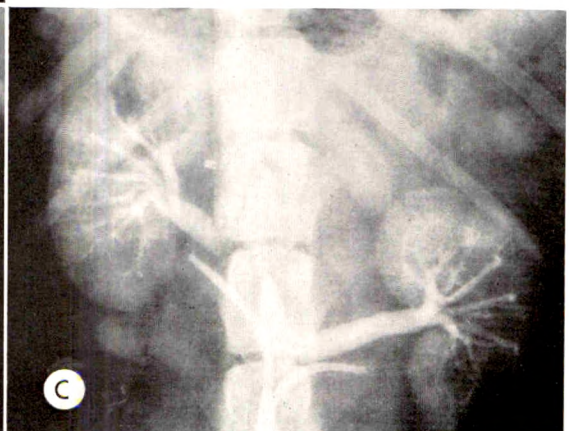
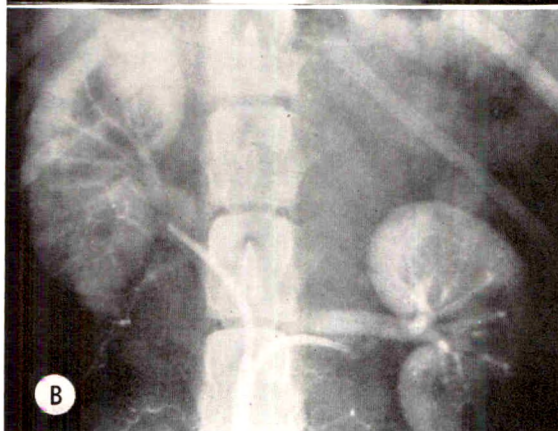


FIG. 3. (*A*) Control simultaneous bilateral selective angiogram with thorotrast, ½ second film, shows normal appearing renal arteries bilaterally. (*B*) Control bilateral angiogram, 2½ second film, shows a good vascular nephrogram on each side with early vein opacification. (*C*) Control bilateral angiogram, 3½ second film, shows good opacification of the renal veins bilaterally.





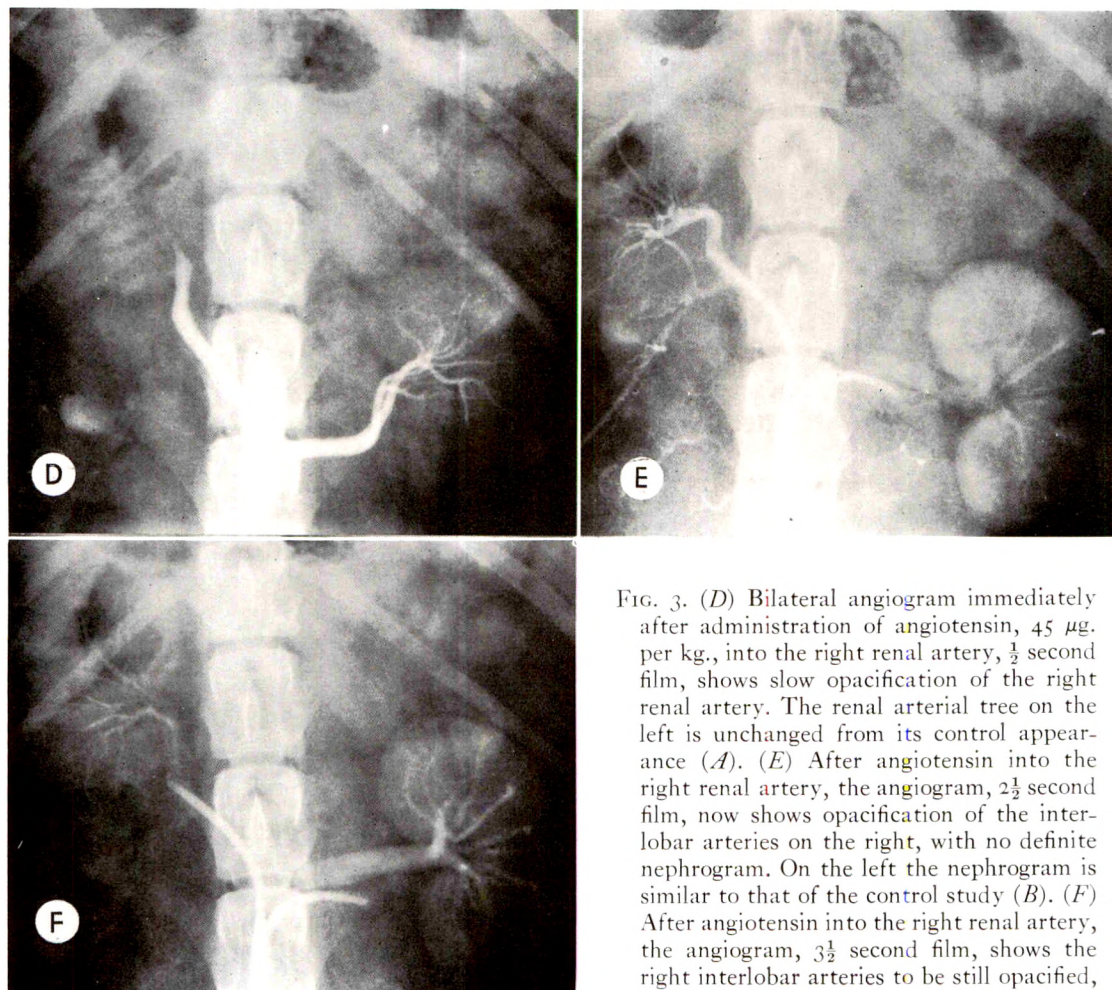


FIG. 3. (D) Bilateral angiogram immediately after administration of angiotensin, 45  $\mu$ g. per kg., into the right renal artery,  $\frac{1}{2}$  second film, shows slow opacification of the right renal artery. The renal arterial tree on the left is unchanged from its control appearance (A). (E) After angiotensin into the right renal artery, the angiogram,  $2\frac{1}{2}$  second film, now shows opacification of the interlobar arteries on the right, with no definite nephrogram. On the left the nephrogram is similar to that of the control study (B). (F) After angiotensin into the right renal artery, the angiogram,  $3\frac{1}{2}$  second film, shows the right interlobar arteries to be still opacified, with only a faint nephrogram. On the left

there is good venous opacification similar to the appearance of the control series (C).

constriction of the main renal artery or its major branches. The interlobar arteries appeared the same size as in the control study, or possibly slightly dilated; the nephrogram intensity was markedly diminished. On the left side, the angiogram showed no change from the control series (Fig. 3, A-F). The angiographic exposures were completed before the onset of the blood pressure rise produced by the angiotensin. Follow-up bilateral angiograms, done 5 minutes later, showed both sides to appear similar, like the control except for slight dilatation of the interlobar arteries and slight slowing of renal circulation bilaterally; the blood pressure had returned to the pre-angiotensin level. At 30 minutes the bilateral angio-

grams had returned completely to normal. These results suggest that angiotensin has a direct action on the renal vessels and that the observed vascular changes are not secondary to changes in the systemic blood pressure.

#### DISCUSSION

Studies of the effects of several vasoconstrictor drugs on renal vascularity in the dog<sup>5,9</sup> have indicated two types of response. As inferred from changes in the renal angiograms, there appear to be a "peripheral" vasoconstrictive effect and a "central" vasoconstrictive effect. By "peripheral" vasoconstriction is meant increased vascular resistance distal to the interlobar arter-

TABLE I  
ANGIOGRAPHIC FINDINGS IN PERIPHERAL AND CENTRAL VASOCONSTRICTIVE EFFECTS

	Size of Main Renal Artery	Size of Interlobar Arteries	Intensity of Nephrogram	Circulation Rate
Peripheral	Increased	Increased	Normal or Diminished	Normal or Diminished
Central	Increased	Diminished	Diminished	Diminished

ies resulting in the following angiographic changes: increased size of the main renal artery, its major branches and the interlobar arteries; normal or diminished intensity of the vascular nephrogram; and normal or slowed passage of the opacified blood from renal artery to renal vein. By "central" vasoconstriction is meant increased vascular resistance at the distal portion of the main renal artery or at the proximal portion of its primary branches. The angiographic changes include: narrowing of the vessel or vessels in the region of the increased vascular resistance; diminution in the size of the interlobar arteries; diminished intensity of the vascular nephrogram; and slowed passage of the opacified blood from renal artery to renal vein. These changes are summarized in Table I.

TABLE II  
PERIPHERAL VASOCONSTRICTIVE EFFECT

Drug	Dose (μg. per kg.)	Rise in Mean Blood Pressure (mm. Hg)
Epinephrine	2 to 5	40-75
Levarterenol	0.5 to 1	15-75
Metaraminol	50 to 100	50-80
Angiotensin	0.5 to 45	50-120

TABLE III  
CENTRAL VASOCONSTRICTIVE EFFECT

Drug	Dose (μg. per kg.)	Rise in Mean Blood Pressure (mm. Hg)
Epinephrine	10 to 60	80-130
Levarterenol	5 to 45	85-110
Metaraminol	200 to 400	80-120

The vasopressors so far tested include: epinephrine, levarterenol, metaraminol and angiotensin. Within the dose ranges evaluated (to produce maximal rise of mean blood pressure by about 120 mm. Hg), the sympathicomimetic drugs have shown both effects related to the dose used. The smaller doses, producing rises in mean blood pressure in the order of 15 to 80 mm. Hg, have shown "peripheral" vasoconstriction (Table II). The larger doses, producing rises in mean blood pressure in the order of 80 to 120 mm. Hg, have demonstrated "central" vasoconstriction (Table III). On the other hand angiotensin, regardless of the dose used, has shown only "peripheral" vasoconstriction (Table II).

SUMMARY

Angiographic study has been made of the effects of angiotensin on the renal vessels of the dog.

All tested doses (0.5 to 45 μg. per kg. intravenously) produced changes in the appearance of the renal vessels suggesting vasoconstrictive action on vessels peripheral to the interlobar arteries.

Angiotensin has a direct action on the renal vasculature.

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## THE VALUE OF NEPHROTOMOGRAPHY IN THE DIFFERENTIAL DIAGNOSIS OF ABDOMINAL MASSES\*

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THE differential diagnosis of abdominal masses is a common clinical and roentgenologic problem. When discovered, such a mass usually requires extensive study to determine etiology, location, size, extent and possible involvement of surrounding tissues and organs.<sup>2,6</sup>

From a roentgenologic viewpoint, evaluation begins with the conventional examinations.<sup>4</sup> A preliminary abdominal roentgenogram often demonstrates a loss of psoas shadows, calcification or the outline of a soft tissue mass. Intravenous pyelography may show deflection of the ureter or displacement, rotation or distortions of a kidney outline and its collecting system. Gastrointestinal studies may demonstrate intrinsic tumors or displacement of the intestine by adjacent masses.

In recent years, more specialized roentgen procedures have aided in the differential diagnosis of abdominal masses. Presacral gas insufflation, retrograde catheter study of the aorta and great vessels, or their combined use have been helpful in the investigation of retroperitoneal masses.<sup>5</sup> Although simultaneous retroperitoneal gas insufflation and aortography have been quite satisfactory, we have been impressed by the information afforded by intravenous aortography and tomography of the abdomen.

Nephrotomography, the sequence of abdominal aortography and tomography, was originally conceived for the differential diagnosis of renal cysts and neoplasm. However, experience has shown its value in retroperitoneal and abdominal diagnostic problems.<sup>1,3</sup>

Nephrotomography, by definition, af-

forded both the vascular delineation of the kidney area and tomographic demonstration of levels of the opacified kidney parenchyma. Modifications of this technique to include extrarenal areas, if the vascular and nephrogram phases are utilized, may be of tremendous value in the localization, definition and diagnostic evaluation of abdominal masses. This has been especially true in differentiating masses adjacent to the kidney silhouette and evaluating their exact relationship to the kidney, *i.e.*, intrinsic or extrinsic to kidney parenchyma. When further roentgen study is necessary, pneumoperitoneum or a retroperitoneal air gas insufflation is performed in conjunction with percutaneous retrograde catheterization.

### TECHNIQUE

An antecubital cutdown is made and a 12 gauge Robb Steinberg needle is placed in the vein. The circulation time is determined with decholin. The needle is then connected via a polyethylene adapter to a Gidlund automatic injector. The injection is made under low pressure 2.2 to 2.4 kg./cm.<sup>2</sup> to ensure a continuous bolus. The actual injection takes from 2 to 3 seconds. Angioconray, 80 cc., is the usual contrast medium and the dosage for the average adult. The rapid cassette changer is loaded with 15 films to allow exposures of 1 per second. The exposures should start from 4 to 5 seconds before the circulation time and continue to at least 5 to 6 seconds after the circulation time. Duration of each exposure is  $\frac{1}{5}$  of a second.

After the 15 exposures of the arterio-

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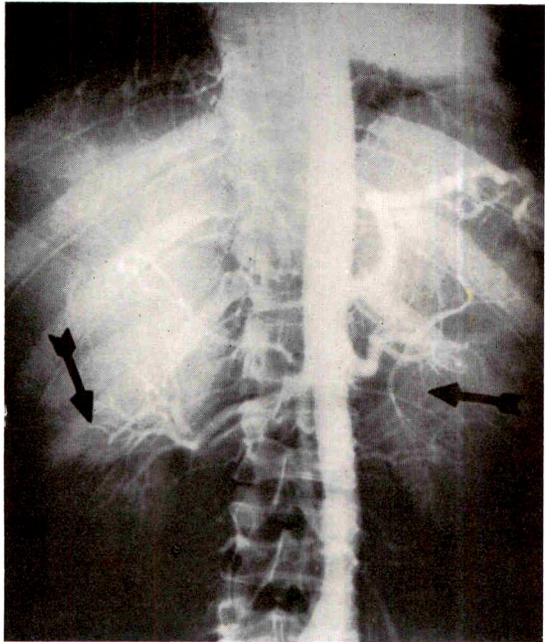


FIG. 1. Case 1. The vascular phase of the nephrotomogram shows that the renal arterial tree is displaced by the larger kidney cysts (arrows).

graphic phase, the table top is returned to normal position so that tomographic exposures can be obtained without delay.

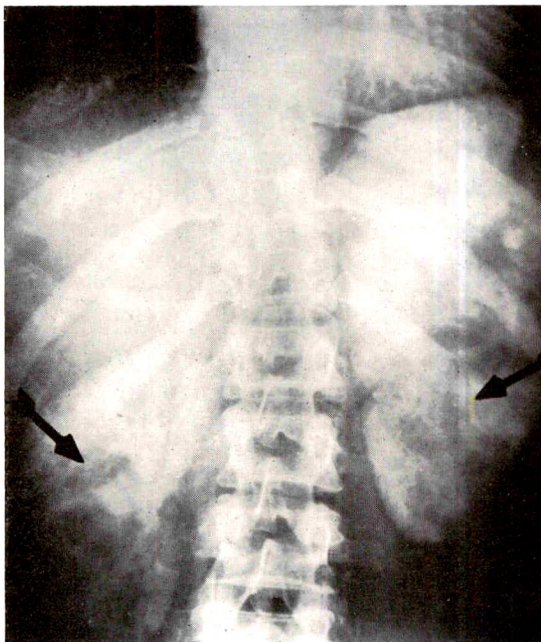


FIG. 2. Case 1. The nephrogram phase shows defects at the cystic areas (arrows).

The following cases illustrate the above technique.

#### ILLUSTRATIVE CASES

CASE 1. The patient was a 28 year old housewife who was referred for intravenous nephrotomography. An intravenous pyelogram, obtained elsewhere, definitely showed one space-occupying mass in the left kidney with a suggestion of perhaps one or two other cysts in this kidney. The right kidney appeared to be completely normal. The patient was asymptomatic. A urinalysis and blood urea nitrogen were normal. The patient's father, however, was noted to have died of renal disease.

An intravenous nephrotomogram showed innumerable small filling defects throughout both kidneys having the appearance of small cysts (Fig. 1, 2 and 3). Within the left kidney, in addition, there were several much larger cystic masses; these were the masses which had been identified on an intravenous pyelogram. On the intravenous pyelogram, almost no distortion of the collecting system of the right kidney was present. The vascular phase of the nephrotomogram showed that the renal arterial tree was intrinsically normal although displacement of branches of the renal arteries by the larger

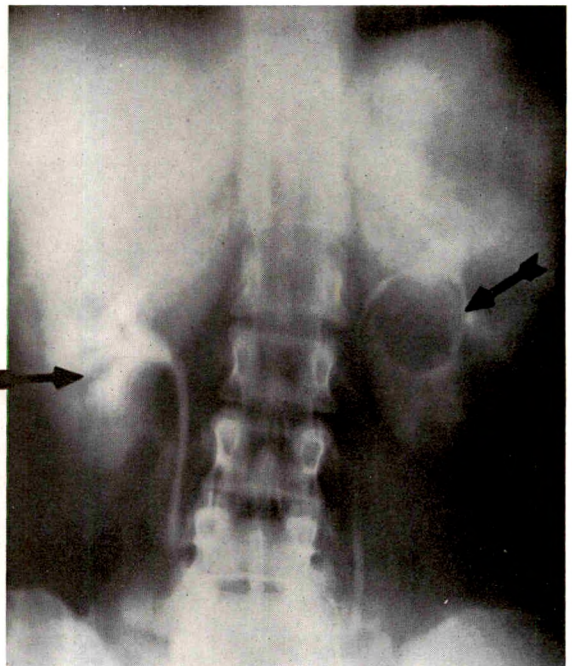


FIG. 3. Case 1. Tomogram helps define the cystic areas (arrows).



cystic masses was present in the kidneys. The diagnosis was polycystic kidneys.

*Comment.* Nephrotomography will often uncover unsuspected renal and extrarenal disease.

CASE II. The patient was a 54 year old Iranian woman who, 6 months prior to admission, had several episodes of hematuria with severe generalized abdominal pain. Several calcium urate urinary calculi were passed at that time. An intravenous pyelogram made 1 week prior to admission showed a mass involving the lower pole of the right kidney. There was no history of weight loss, anorexia, nausea or vomiting. Physical examination revealed a blood pressure of 160/88; pulse of 84 and regular; respiration 18; temperature 99° F. Examination was negative other than the presence of a hard nontender fixed mass in the right upper quadrant. Multiple urinalyses showed 25 to 30 red blood cells per high power field with occasional white blood cells. A catheterized urine specimen revealed *E. coli*.

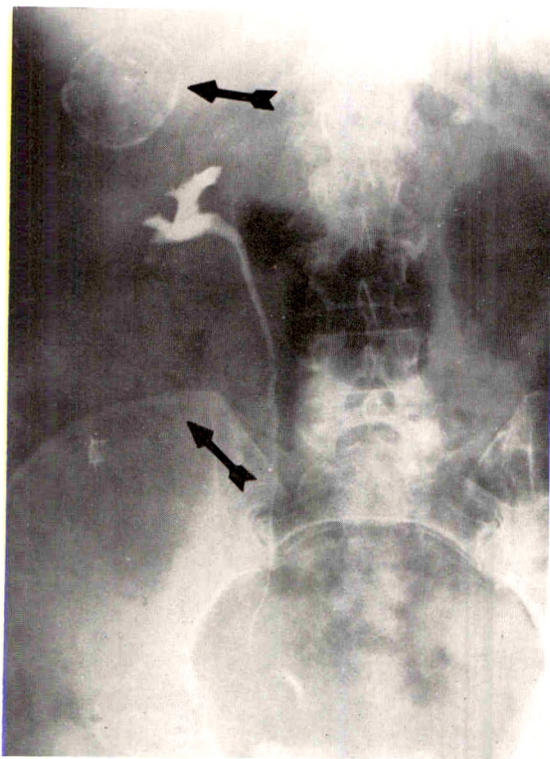


FIG. 4. Case II. Retrograde pyelogram shows a large mass replacing the lower pole of the right kidney (lower arrow). A calcified cyst is noted in the liver area (upper arrow).

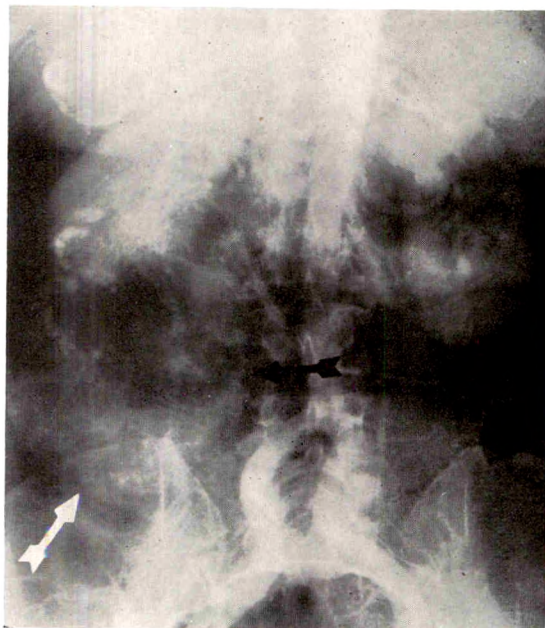


FIG. 5. Case II. The arterial phase of nephrotomography shows that the renal mass is highly vascular with a malignant pattern (arrows).

A plain roentgenogram of the abdomen prior to intravenous nephrotomography showed a large mass replacing the lower pole of the right kidney extending inferiorly. The mass measured

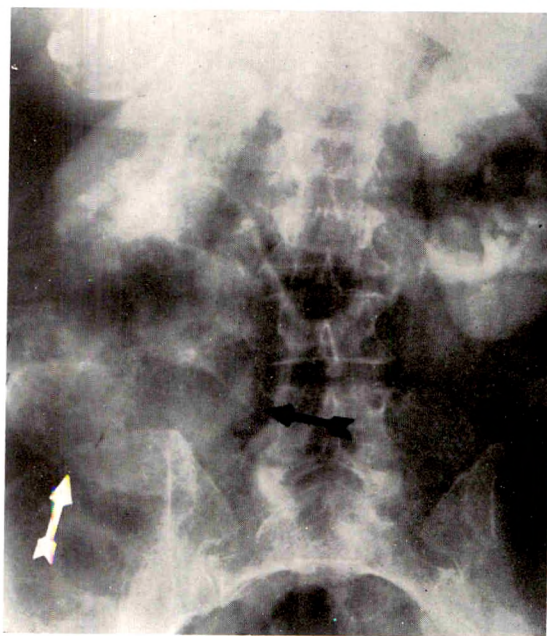


FIG. 6. Case II. Late in the arterial phase, a tumor stain is seen (arrows).





FIG. 7. Case II. Tomogram defines the extent of the tumor (arrows) and the level of the calcified liver cyst.

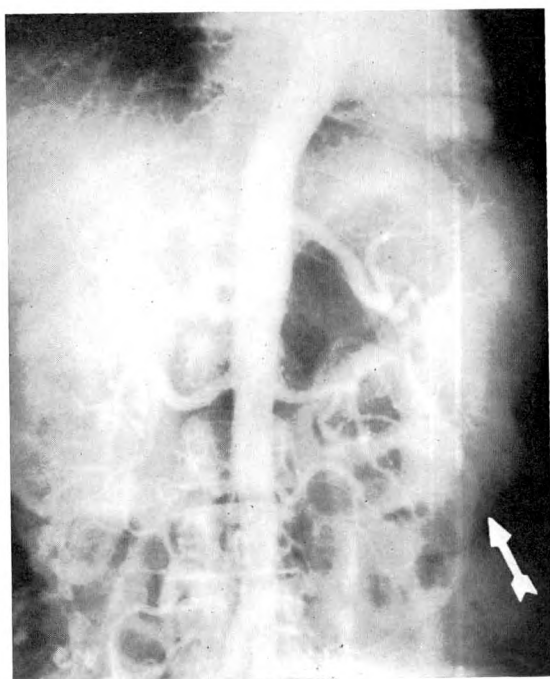


FIG. 8. Case III. Arterial phase of a nephrotomogram is normal. The arrow marks the lower tip of the enlarged spleen.

approximately 7 cm. in diameter. In addition there was a calcified rounded mass in the right upper quadrant which had the typical roentgenographic appearance of an echinococcus cyst of the liver (Fig. 4). The arterial phase of nephrotomography showed that the renal mass was highly vascular and that the vascular pattern was that of a malignant tumor with marked irregularity of vessels, numerous arteriovenous fistula effects and a very marked increase in vascular channels (Fig. 5). The tomographic nephrogram phase of the study showed again that the tumor was highly vascular but that there were irregular areas of nonvascularity, suggesting either cystic areas or necrotic areas within the tumor (Fig. 6 and 7).

A right nephrectomy was performed and showed that the lower pole of the right kidney was completely replaced by a large renal tumor, measuring  $10 \times 15 \times 12$  cm. There was no apparent involvement of the renal pedicle or of the adjacent lymph nodes or perirenal fat. The histologic report was "adenocarcinoma of the kidney, clear cell type."

*Comment.* Arteriography and tomography in this case defined the limits of renal pathology. Tomography revealed the level of the echinococcus cyst.

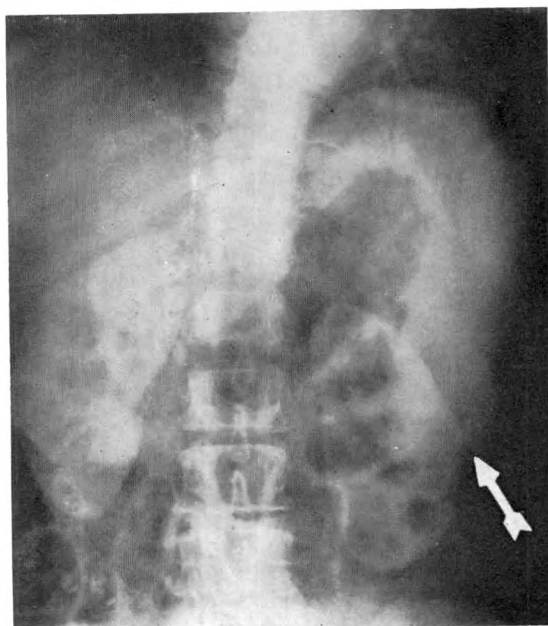


FIG. 9. Case III. Nephrogram phase well demonstrates the splenic tissue (arrow).

CASE III. The patient was a 73 year old Haitian born widow who presented with a chief complaint of increasing abdominal girth of 8 days' duration. She had been in excellent health until 2 months prior to admission when she noted increasing fatigue. Eight days prior to admission she was found by her local physician to have hepatosplenomegaly. Upon questioning, the patient stated that she had frequent nose bleeds during the past winter; there was no other evidence of bleeding. She had had a weight loss of about 40 to 50 pounds but stated that this had occurred over a 4 year period, during which time she had dieted.

Physical examination revealed a thin pleasant French-speaking Negro female who appeared much younger than her stated age of 73. The blood pressure was 160/80. Other vital signs were within normal limits. The sclerae were not icteric. The liver was down 3 fingerbreadths at the mid-clavicular line; it was non-tender. There was a mass in the left upper quadrant which was 4 fingerbreadths down from the costal margin and was slightly tender; this was felt to represent the spleen.

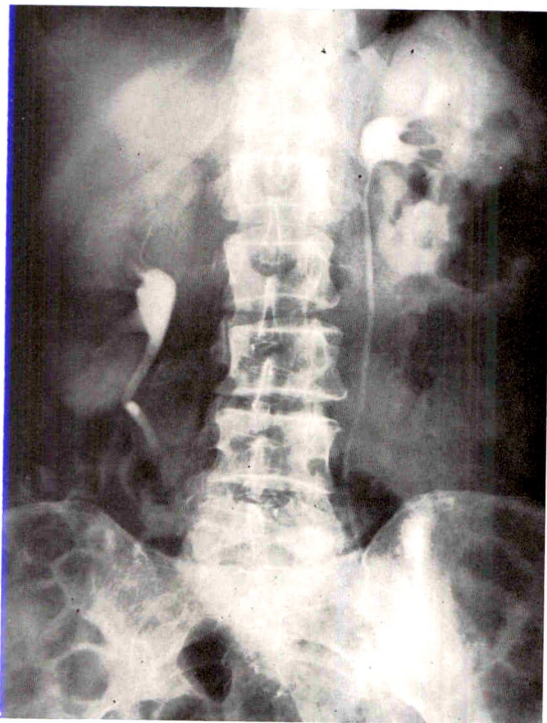


FIG. 10. Case IV. Intravenous pyelogram shows the right kidney to be normal. The left kidney is displaced upward and laterally.

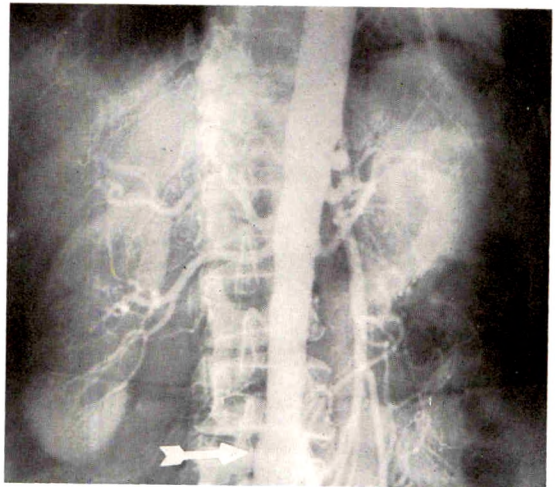


FIG. 11. Case IV. Arteriogram shows a normal left renal artery displaced upward with the kidney and an aneurysm of the lower abdominal aorta (arrow).

An intravenous pyelogram showed a normal right kidney, but some irregularity of the calyces within the mid-portion of the left kidney was noted. An intravenous nephrotomogram showed both kidneys to be normal. No abnormal renal masses were identified. This study well demonstrated the clinically recognized splenomegaly (Fig. 8 and 9).

*Comment.* Nephrotomography was performed to better evaluate the left kidney. In so doing, the enlarged spleen was clearly defined. Nephrotomography can be helpful in documenting hepatosplenomegaly and its effect on the kidneys and surrounding region.

CASE IV. The patient was a 65 year old white female admitted to the hospital with a presenting complaint of diffuse abdominal pain for 4 weeks. The pain was unrelated to position, food or activity. There was no melena and no weight loss. The patient had had a subtotal gastrectomy and splenectomy in 1962 for carcinoma of the stomach. Physical examination showed the abdominal scar from the previous surgery. There was some suggestion of a firm nonfixed mass in the right lower quadrant of the abdomen.

An intravenous pyelogram showed the right kidney to be normal in size, position and configuration (Fig. 10). The left kidney was displaced upward and laterally. There was some



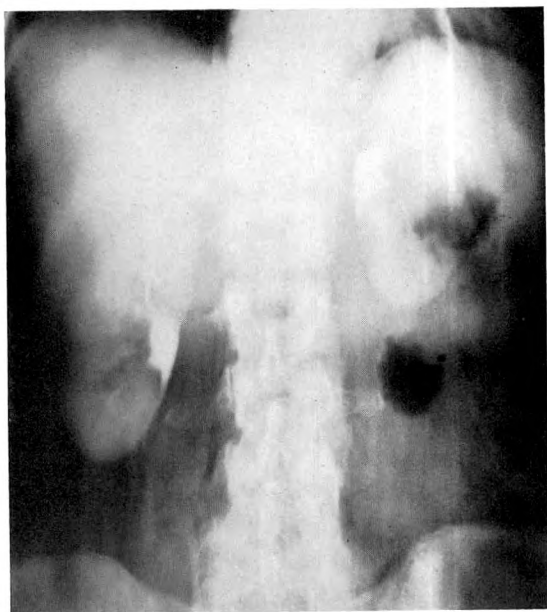


FIG. 12. Case IV. Tomogram (with arteriography) rules out the presence of a mass.

distortion of the upper and middle calyces of the left kidney.

Because of the findings on the intravenous pyelogram, intravenous nephrotomography was performed. There was no evidence of renal tumor. The displacement and rotation of the

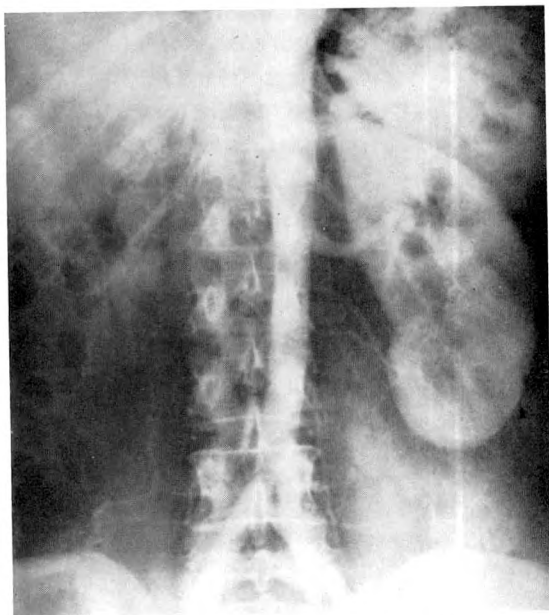


FIG. 13. Case V. Arteriogram shows absence of right renal artery.

left kidney was thought to be secondary to the known gastrectomy and splenectomy and the distortion of the collecting system was in turn thought to be secondary to the rotation of the left kidney. An incidental finding on the nephrotomogram was the presence of a small aneurysm in the lower abdominal aorta (Fig. 11).

*Comment.* The possibility of an abdominal tumor displacing the kidney upward was effectively ruled out (Fig. 12). The unsuspected aortic aneurysm was demonstrated just proximal to the aortic bifurcation.

CASE V. The patient was admitted to the hospital with a history of postmenopausal vaginal bleeding for 1 year. The patient had no symptoms referable to the urinary tract and no history of previous urinary tract disease.

Physical examination was completely within normal limits except for the pelvic examination which showed a pliable lesion of the cervix, bleeding readily on contact. This proved to be a Stage II squamous cell carcinoma of the cervix, which

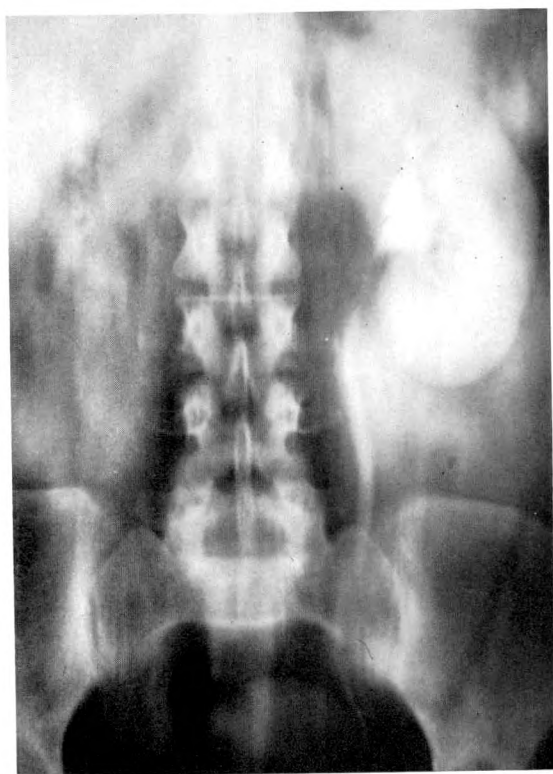


FIG. 14. Case V. Tomogram shows definitely the absence of a functioning right kidney.

was treated by a combination of intracavitary and external radiation therapy.

An intravenous pyelogram made in the investigation of the above lesion showed a large left kidney which functioned normally. There was, however, no evidence of an outline of a right kidney or of excretion by a right kidney. The cystoscopist was unable to locate an orifice for the right ureter. An intravenous nephrotomogram demonstrated a large left kidney. On the right side no renal artery was visualized and no evidence of an outline of the right kidney was definitely noted (Fig. 13). Tomographic studies showed a small density posteriorly at the level of the psoas outline which might have been a small nonfunctioning right kidney (Fig. 14).

The patient was last seen in May, 1965 at which time she was asymptomatic and there was no evidence of recurrent uterine disease.

*Comment.* Nephrotomography definitely showed the absence of the right renal artery.

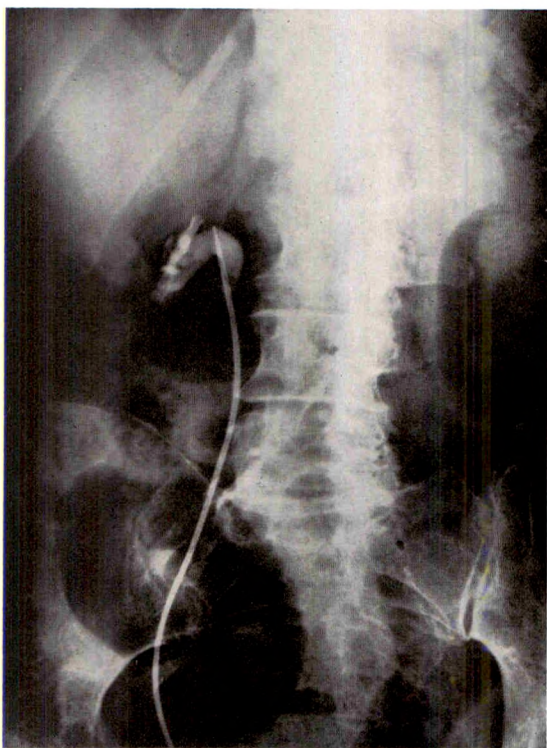


FIG. 15. Case VI. Right retrograde pyelogram shows a small kidney and collecting system with minimal caliectatic changes.

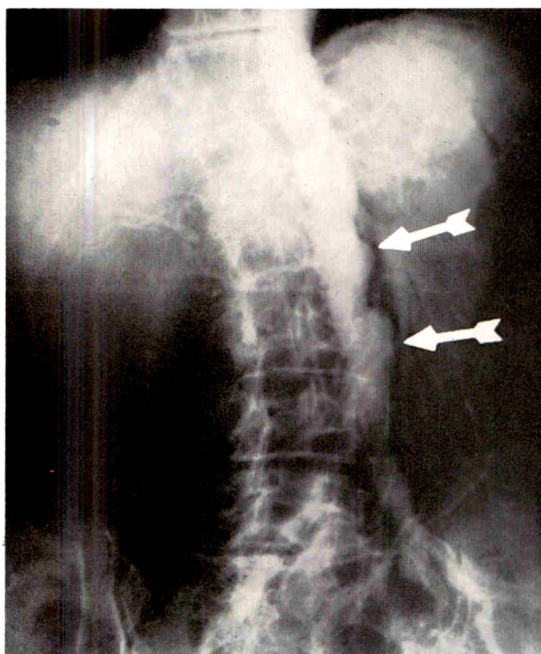


FIG. 16. Case VI. The arterial phase of nephrotomography shows extreme arteriosclerotic changes in the abdominal and iliac vessels with aneurysm formation (arrows).

**CASE VI.** This was the first hospital admission of a 69 year old white female widow, former resident of Singapore, who entered the hospital with the chief complaint of dyspnea, episodes of substernal crushing pain and high blood pressure. Prior to her present illness, the patient was in good health. She was known to have had high blood pressure since 1955 with initial readings of 170/90.

Physical examination revealed a well developed, sallow white female in slight respiratory distress. The blood pressure was 200/100; pulse 92 and regular; and respirations 22. The chest was slightly dull at both bases. There were inspiratory rales at both posterior bases, up to the angle of the scapula. The heart was irregular; there were no murmurs. There was 4+ pitting, pedal, pretibial and sacral edema.

Following an intensive medical regimen, she was slowly ambulated and had no difficulty with breathing or further episodes of paroxysmal nocturnal dyspnea. She was noted to have a urinary tract infection which responded well to Furadantin and cleared completely. Because of the urinary tract infection, intravenous pyelography was performed. No function was seen on the right side. A right retrograde pyelo-



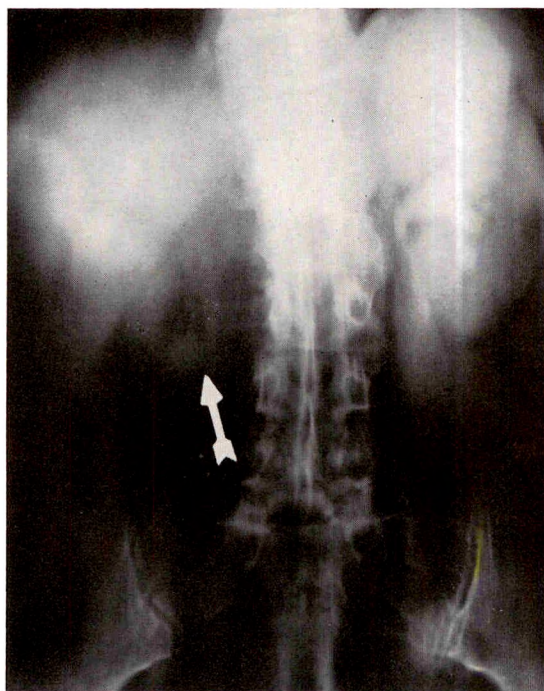


FIG. 17. Case VI. Tomogram better outlines the small right kidney (arrow).

gram (Fig. 15) showed a small collecting system with minimal caliectatic changes. Nephrotomography showed extensive arteriosclerotic changes in the abdominal aorta and iliac vessels with aneurysm formation (Fig. 16). A small right kidney with an atrophic renal artery was seen. Tomograms showed the small kidney with suggestions of some calcifications (Fig. 17).

*Comment.* In the process of evaluating the right kidney, considerable unsuspected arterial disease with aneurysm formation was uncovered.

CASE VII. A 54 year old white male was admitted with a history of intermittent epigastric pain for approximately 1 year. The pain was more pronounced in the morning and was partially relieved by alcohol and food. Three months prior to admission, he had been told by his physician that he was anemic. The patient had been placed on iron medication.

Physical examination showed a thin white male in no distress. Vital signs were normal. Examination of the abdomen disclosed an enlarged liver with dullness, extending 1 to 2 fingerbreadths below the right costal margin. No masses were felt.

Laboratory studies showed a severe anemia and multiple stool guaiac tests ranged from negative to 1 to 2+. A bone marrow was normal. A string test with fluorescein dye showed no evidence of active gastrointestinal bleeding at the time of the study.

A gastrointestinal series showed a constrictive lesion involving the distal duodenum in the region of the ligament of Treitz (Fig. 18). Gross destruction of normal mucosal markings was present. The changes were thought to be consistent with an intrinsic neoplasm. Nephrotomography via the retrograde route was performed together with a presacral retroperitoneal gas insufflation study. A mass inferior to the left kidney was demonstrated, measuring approximately 10×7 cm. Numerous branches of the superior and inferior mesenteric arteries supplied the tumor (Fig. 19, 20 and 21).

At surgery, a large ulcerating mass was found in the terminal duodenum just beneath the ligament of Treitz and the superior mesenteric vessels. The mass measured approximately 8 cm. in length. Firm nodules were felt in the liver and were present in several lymph nodes in the area. The lesion was a primary carcinoma



FIG. 18. Case VII. Gastrointestinal study shows a constrictive lesion involving the distal duodenum in the region of the ligament of Treitz (arrow).



of the terminal duodenum with hepatic metastases.

*Comment.* A combination of catheterization, presacral air insufflation and tomography can be extremely helpful in a selected case.

**CASE VIII.** The patient was a 45 year old male who was first seen in the clinic in January 1965 with a complaint of mid-epigastric burning pain for over a year. At a previous hospitalization, the patient had been told that he had an ulcer. Pain was partially relieved by food and mild medication. A gastrointestinal series showed irritability of the duodenal cap with hypersecretion in the stomach. A large soft tissue mass was demonstrated in the left upper quadrant; this mass caused some extrinsic pressure on the stomach. An intravenous pyelogram showed a normal right renal outline, but the left renal silhouette was only faintly visualized. Intravenous nephrotomograms (Fig. 22) showed normal renal arteries bilaterally.

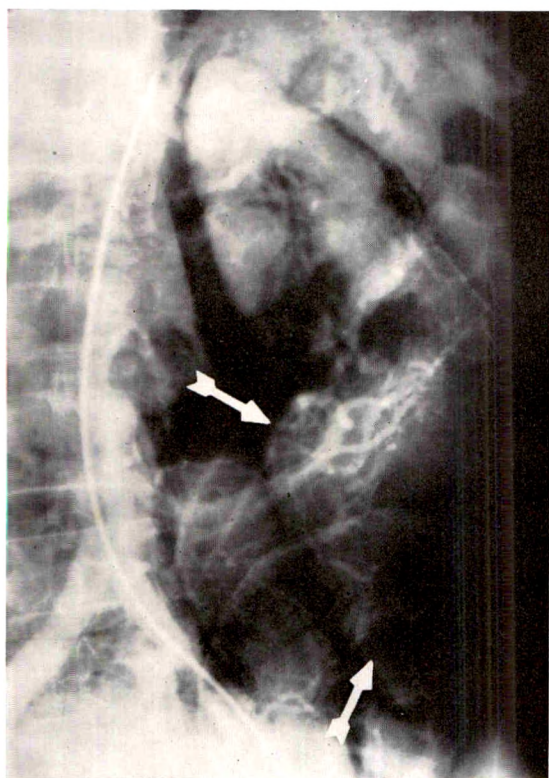


FIG. 19. Case VII. Nephrotomography and retroperitoneal gas insufflation show a mass inferior to the left kidney (arrows).

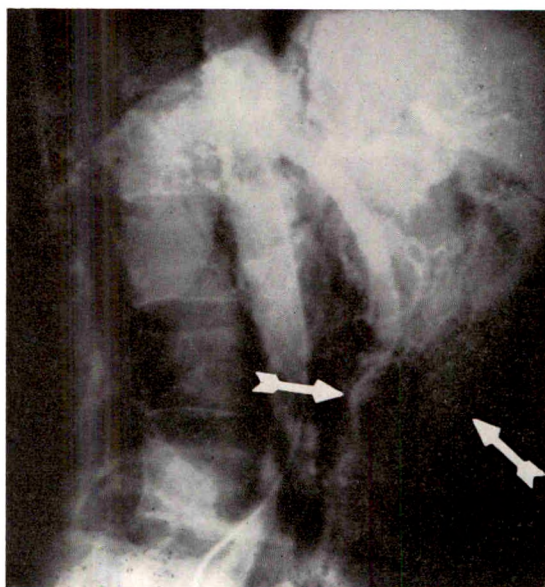


FIG. 20. Case VII. Lateral arteriogram shows abnormal vessels (arrows).

The right kidney was normal. The left kidney appeared to be generally enlarged. There was no renal mass. There was, however, a large soft tissue density overlying the left renal shadow which appeared to be anterior to the



FIG. 21. Case VII. Tomographic study shows the mass not to be in the retroperitoneal space.



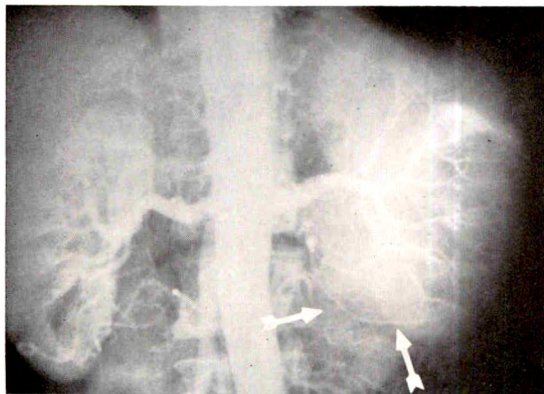


FIG. 22. Case VIII. Intravenous nephrotomogram shows normal renal arteries bilaterally. An overlying large soft tissue mass is seen in the lower left kidney area. Note vessels bowed around the mass (arrows).

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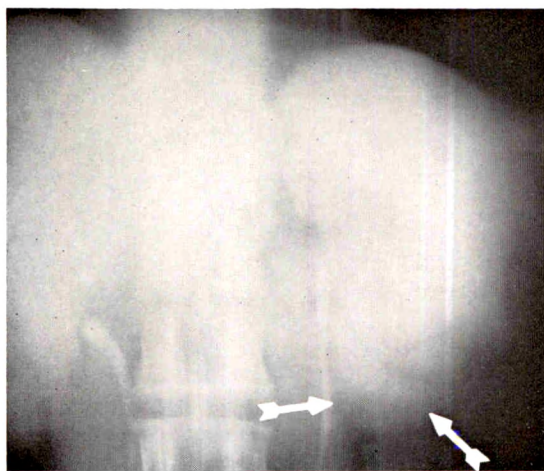


FIG. 23. Case VIII. Tomogram in the frontal position defines the lower tip of the mass (arrows).

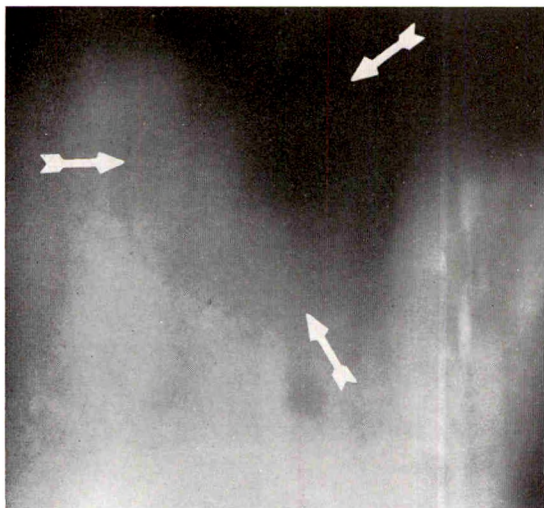


FIG. 24. Case VIII. Lateral tomogram shows the mass anterior to the kidney (arrows).

left kidney and was thought to represent the spleen (Fig. 23). Lateral tomograms showed the mass anterior to the kidney (Fig. 24).

At surgery, a large cyst measuring approximately 15 cm. in greatest diameter was found in the spleen. The cyst was surrounded by an intense inflammatory reaction with adhesions. The pathology was that of a thick walled fibrocystic spleen containing old clotted blood which was thought to be an old splenic hematoma.

*Comment.* Lateral tomograms following arteriography were most helpful in defining the relationship of the cyst to the normal left kidney.

CASE IX. The patient was well until the day of admission when he experienced severe abdominal and back pain that radiated to the right lower quadrant and then to the groin. He vomited immediately following the pain, and, when seen by his local physician, was noted to have a blood pressure of 66/0. The patient ap-

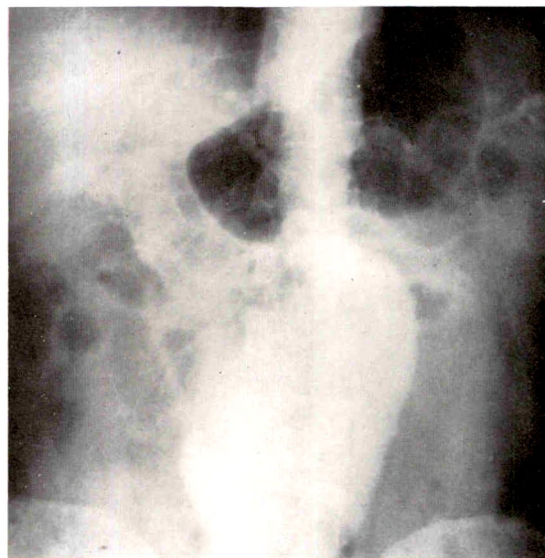


FIG. 25. Case IX. Arteriogram shows a large abdominal aneurysm extending from the renal arteries to the aortic bifurcation.



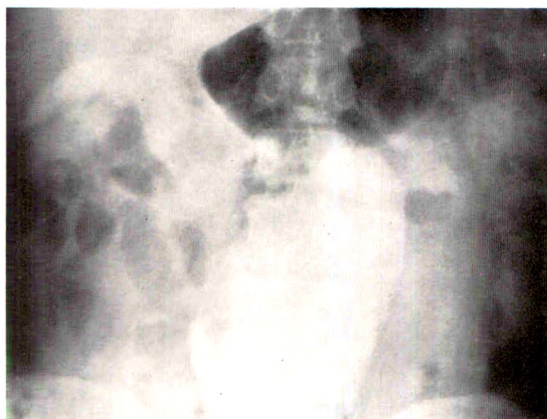


FIG. 26. Case IX. Late in the arterial phase, contrast material pools in the aneurysm.

peared in shock and was immediately admitted to the hospital.

Physical examination revealed a patient in acute distress with pain in the lower abdomen, somewhat cyanotic, cold, and lying quietly in bed. Severe right lower quadrant abdominal tenderness with abdominal distention was present. Pulses appeared to be intact. The clinical impression at that time was a vascular accident of the abdomen, possibly a ruptured aneurysm or mesenteric thrombosis. Arteriography and tomography were performed on the day of admission. A large abdominal aortic aneurysm extending from the renal arteries to the bifurcation of the aorta was found (Fig. 25 and 26). Tomographic studies following aortography demonstrated a large surrounding mass, un-

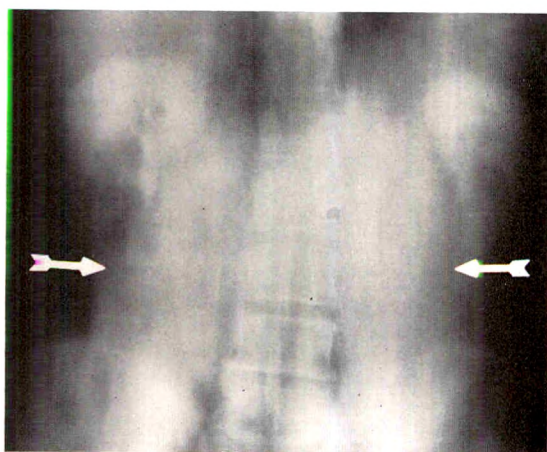


FIG. 27. Case IX. Tomogram following arteriography demonstrates the large surrounding hematoma (arrows).

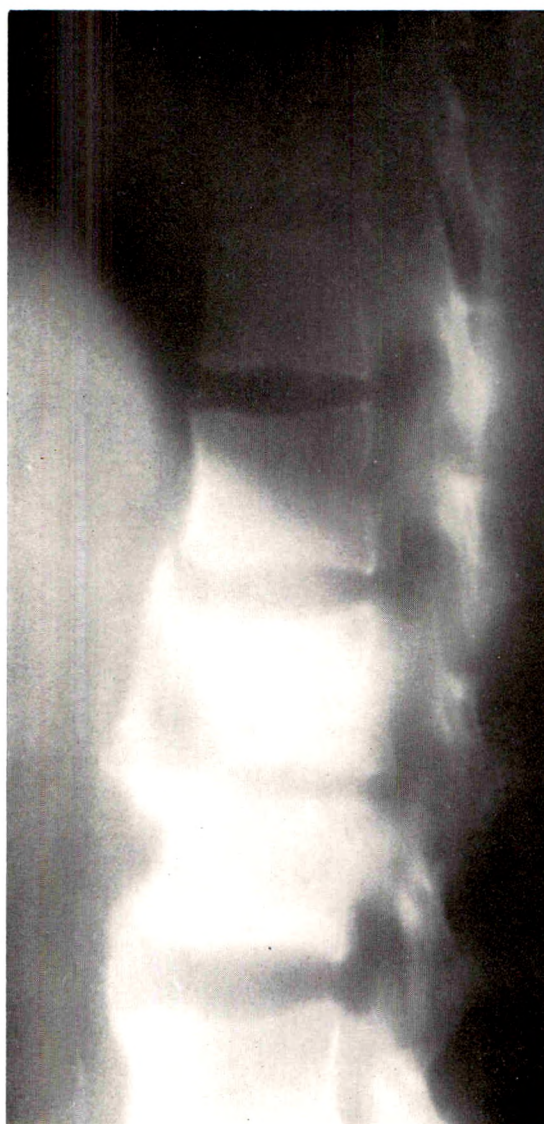


FIG. 28. Case X. Lateral lumbar roentgenogram shows definite decrease in the intervertebral space between L2-L3 with areas of destruction in both vertebral bodies, sclerotic changes and marked spurring.

doubtedly representing hematoma from the leaking aneurysm (Fig. 27). Both kidneys were displaced laterally and the renal arteries were displaced upward but the nephrogram effect showed functioning normal appearing kidneys.

The roentgenographic impression was a large aorta aneurysm with surrounding hematoma due to perforation with displacement of renal arteries and kidneys. An operation was performed on the same day with removal of the aneurysm and replacement with a dacron graft.



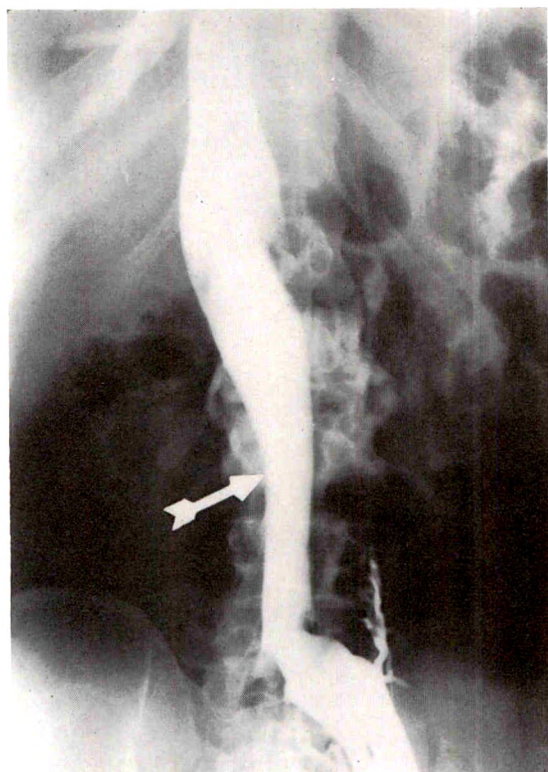


FIG. 29. Case x. Inferior vena cavagram shows a large soft tissue mass in the lower abdomen causing definite extrinsic pressure on the vena cava (arrow).

At operation, a large hematoma surrounding the perforated aneurysm was noted. Postoperatively the patient did well.

**CASE X.** This was the first hospital admission of this 33 year old Negro male who entered with the chief complaint of back pain. The patient gave a history of back injury in the past following a fall. Six months prior to this admission, roentgenograms taken of the mid-back showed "damage" to the back. Physical examination was unremarkable except for some hyperextension of the back with definite pain localized to the area of L2 and L3. Pressure over the L2 spinous area elicited considerable pain.

An intravenous pyelogram showed definite decrease in the intervertebral space between L2 and L3, with areas of destruction in both vertebral bodies, sclerotic changes and marked spurring (Fig. 28). The right psoas margin was obliterated and a soft tissue mass was seen in the right lower quadrant. The impression at this time was tuberculosis versus osteomyelitis

of the 2nd and 3rd lumbar vertebrae. The soft tissue mass was thought to be a psoas abscess.

Intravenous aortography, inferior vena cavagraphy and nephrotomography were performed. A large soft tissue mass in the lower abdomen caused definite extrinsic pressure on the inferior vena cava (Fig. 29). The abdominal aorta, right kidney and superior mesenteric artery were also pressed upon (Fig. 30 and 31). There was no evidence of obstruction of the superior mesenteric artery.

Sclerotic destructive changes in the lumbar spine involving L2 and L3 were again noted. The large mass extended down into the pelvis where it caused slight extrinsic pressure on the superior margin of the bladder. The mass remained fairly avascular throughout the aortographic study. The appearance of the mass and its vascularity did not suggest neoplasm. The impression at this time was a large psoas abscess causing extrinsic pressure on vessels as described above.

Biopsy of the 2nd and 3rd lumbar vertebrae

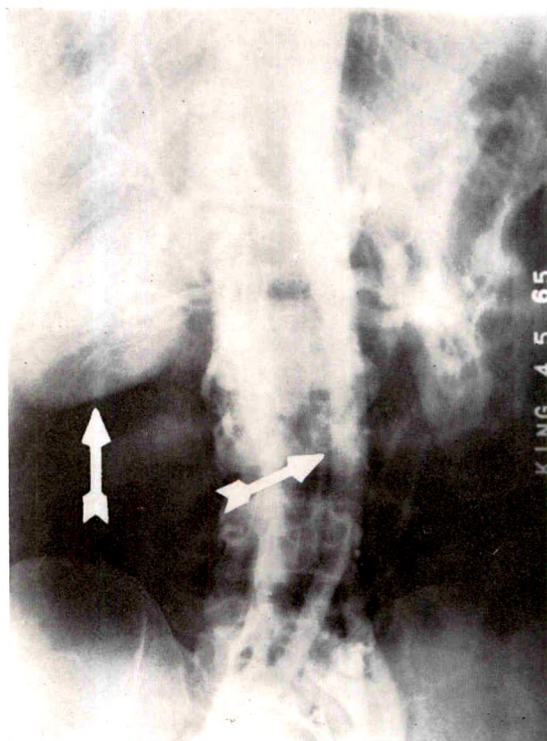


FIG. 30. Case x. During the arterial phase of the injection the abdominal aorta, the right kidney and the superior mesenteric artery are also pressed upon (arrows).



FIG. 31. Case x. Tomogram defines the borders of the relatively avascular mass (arrows).

revealed granulomatous inflammatory tissue of tuberculous etiology.

#### SUMMARY

With the rapid development in experience, contrast material and equipment, a relatively complicated series of studies have gradually slipped into the realm of conventional radiology. Never before has so much reliable information been available

with so little risk and discomfort to the patient. Many believe that it is no longer acceptable to know only that an abnormal mass exists in the abdomen, but rather, that our obligation is to define its relationship to other vital organ systems, to evaluate its blood supply and, to the greatest degree, to define its anatomic characteristics.

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## AN IMPROVED, SIMPLE TECHNIQUE OF LYMPHANGIOGRAPHY

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THE success of a lymphangiographic examination often depends on the practical aspects. Experience shows that in order to obtain lymphangiograms of diagnostic quality, a good and simple technique is necessary. Because of the long duration of the examination and its consequent discomfort to the patient, failures and unnecessary protraction of the examination should be avoided as much as possible.

A very important part of the technique of lymphangiography is the puncture of the lymph vessel, and yet this is seldom described accurately and in detail in the literature. A quick and simple puncture, without the risk of perforation, is the primary requisite. With this in mind, a simple technique was developed in our department which in practice has proved to meet the requirements of the routine examination. Since the preparation of the patient and the injection of the blue dye do not appear to present any difficulties, only the surgical-technical part of the examination is discussed.

### METHOD

After local anesthesia (2 per cent novocaine) of the proximal part of the dorsum of the foot, or the back of the hand, a longitudinal incision of 1 to 1.5 cm. is made in the skin just above the dye colored lymphatic vessel. The incision should not be made too deep, because the subcutaneous lymphatics run superficially. With a pair of fine forceps, the lymphatic vessel is carefully dissected free from the subcutaneous fat

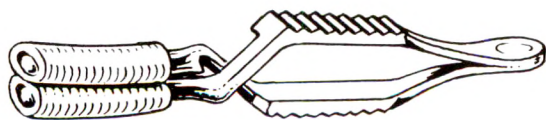


FIG. 1. Special clamp (3.8 cm. long) with rubber-covered tips.

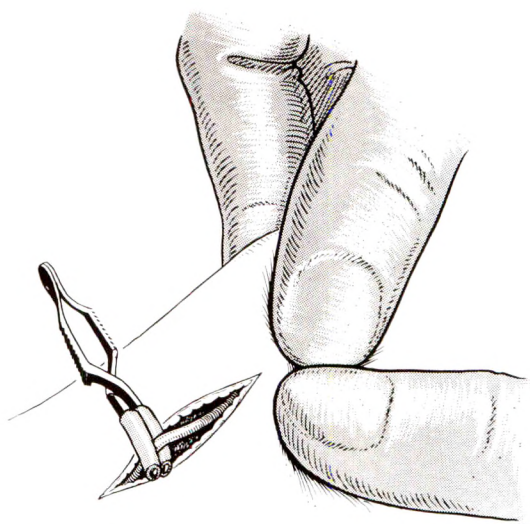


FIG. 2. With the clamp in place, the foot or hand is massaged from distally upward, thus obtaining a distention of the lymphatic vessel to as much as 4 times its normal caliber.

tissue. In relation to the very small diameter of the vessel (0.2 to 0.8 mm.), its wall is very strong and dissection from the adjacent tissues is easy. A special clamp (3.8 cm. long) with rubber-covered tips is placed proximally on the vessel (Fig. 1). In this manner the vessel is protected from injury. The foot or hand is then massaged from distally upward (Fig. 2), whereby a slight, local lymph congestion and distention of the lymphatic vessel are obtained. This distention may cause the vessel to be as much as 4 times the caliber of a normal one.

At this point, the lymphatic vessel can be punctured. However, the vessel is apt to roll from side to side and its posterior wall may easily be perforated. Whenever the vessel is damaged or the needle has to be withdrawn for any reason, the advantage of the induced dilatation is lost and the vessel cannot be punctured again. Another lymph vessel needs to be found and the procedure has to be started all over again. This is not

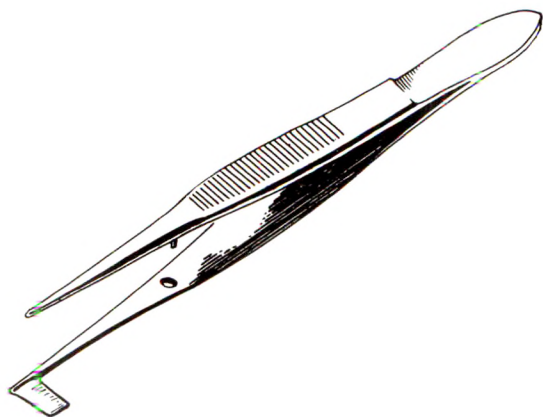


FIG. 3. Special forceps (11.7 cm. long) with smoothly polished inner surfaces and a small metal plate attached at one tip which is placed underneath the lymphatic vessel so as to prevent its injury.

only time-consuming but also very unpleasant to the patient. To prevent such an occurrence, a special forceps (11.7 cm. in length) (Fig. 3) has been devised. The lymphatic vessel is stretched with the forceps, the ends of which are smoothly polished on the inner side so as not to damage the vessel. To one of the tips of the

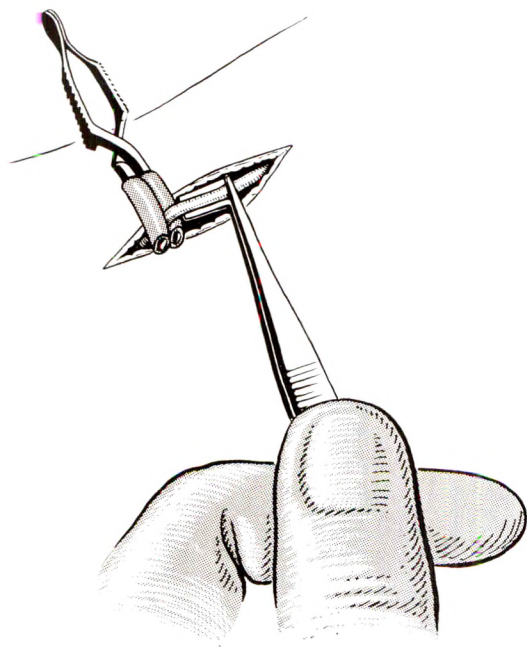


FIG. 4. The lymph vessel is stretched between clamp and forceps and fixed on the plate, making puncture easier.

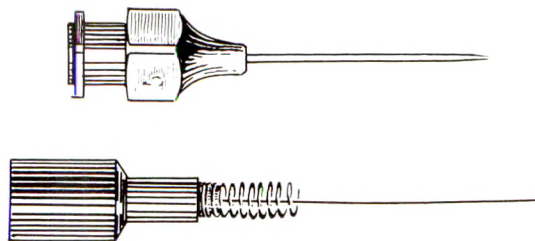


FIG. 5. Special puncture needle, consisting of 2 parts: a smooth, blunt, hollow needle (3.7 cm. long and 0.5 mm. in diameter for the foot, and 0.4 mm. for the hand); and a sharp pointed stylet (4.5 cm. long) with a small spring at the place where the stylet locks into the needle.

forceps a small metal plate has been attached, which is placed underneath the vessel. When the lymph vessel is stretched, it becomes fixed on the plate (Fig. 4), making the puncture much easier.

A special puncture needle is employed (Fig. 5). It was felt that a blunt needle rather than a sharp one would prevent perforation of the lymphatic vessel during puncture and injection. Therefore, a puncture needle was devised which consists of two parts: a smooth, blunt, hollow needle (3.7 cm. in length and 0.5 mm. in diameter for the foot and 0.4 mm. for the hand); and a sharp pointed stylet (4.5 cm. in length).

At first, a needle was used which had a smooth junction with the stylet, but this had the disadvantage that the posterior wall of the vessel could be perforated unnoticed. The needle now employed and which has proved successful for quite some time has a visible junction with the stylet and the end of the stylet protrudes only 0.5 mm. from the needle. A small spring is attached at the place where the stylet locks into the needle. The needle is held between the thumb and the forefinger, while the head of the stylet with the spring is pressed with the third finger (Fig. 6).

The lymphatic vessel is punctured with the slightly protruding, sharp point of the stylet. As the blunt end of the needle reaches the vessel wall, a slight resistance is felt, indicating that the position is satisfactory and the protruding point of the stylet cannot perforate the posterior vessel



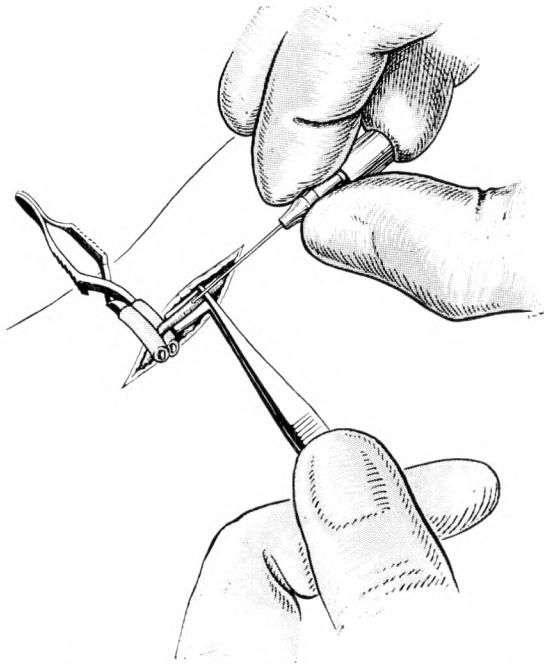


FIG. 6. The needle is held in place between the thumb and the forefinger, while the head of the stylet with the spring is pressed by the third finger. The lymphatic vessel is punctured with the slightly protruding, sharp point of the stylet.

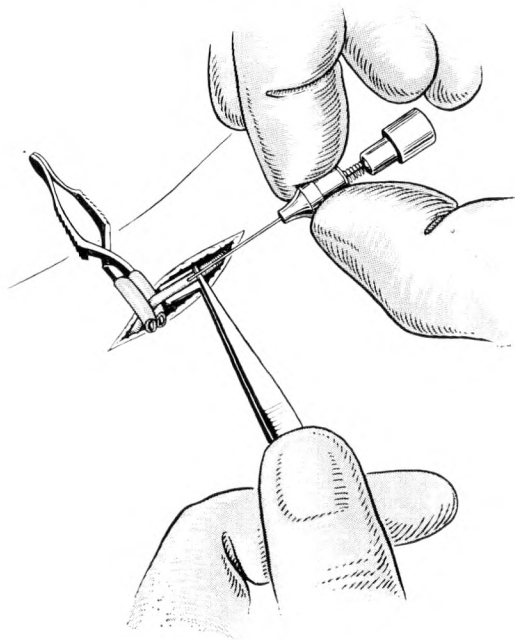


FIG. 7. After the needle is advanced by a rotating movement a few millimeters in the lymphatic vessel, the third finger releases the stylet, thereby releasing the spring, and the stylet shoots back.

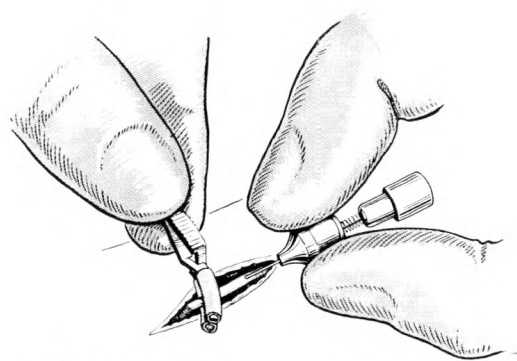


FIG. 8. The blunt, hollow needle is slid into the lymphatic vessel, the clamp is removed and placed on the needle and the vessel to prevent back sliding of the needle.

wall. The needle is now held horizontally and with a rotating movement is advanced a few millimeters farther into the lymph vessel. The third finger releases the stylet (Fig. 7), thereby releasing the string, and the stylet shoots back.

The blunt, hollow needle is slid into the lymphatic vessel, the clamp is removed and placed on the needle and vessel (Fig. 8) to prevent back sliding of the needle. In our experience, no leakage ever occurred when the contrast medium was injected. The

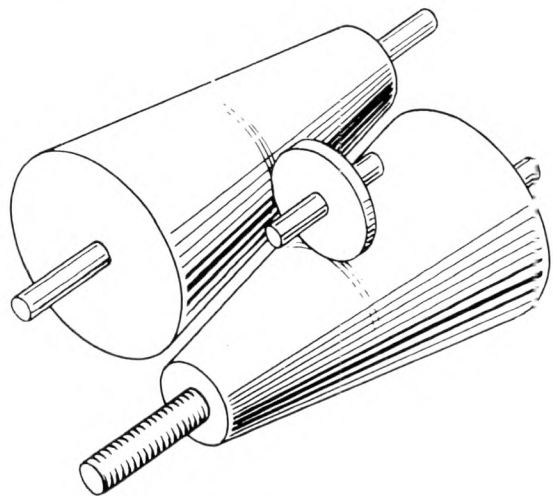


FIG. 9. Specially designed injection apparatus operated by an electromotor. Its gear consists of 2 upright conical rolls which, by means of an adjustable intermediate disk, make it possible to change the speed of injection at any time during the injection.

needle is then fixed to the foot or hand by means of tape.

A polythene catheter connects the needle with a 10 ml. injection syringe with a Luer Lock connection. Since, due to bluntness of the needle, perforation of the vessel wall cannot occur, absolute immobilization of the involved extremities during the injection of contrast medium is no longer necessary. The choice of contrast medium (aqueous or oily) and the method of injection are less important. The injection may be done by hand, or with the help of a motor or weights, as long as it is done slowly and under constant pressure. When oily contrast media are used, the injection speed should not exceed 1 ml. per 5 minutes so as to prevent complications such as rupture of the lymphatics or lymph nodes in the inguinal or axillary region. I used a specially designed injection apparatus operated by an electromotor. Its gear consists of 2 upright conical rolls (Fig. 9), making it possible, by means of an adjustable intermediate disk, to change the speed of injection at any time, even during the injection. Also, this gearbox has the advantage that "a slip" automatically occurs if the counterpressure in the lymphatic system gets too high.

At the end of the injection, the small wound is sutured. An excellent and more simple method is the application of tape strips\* to achieve continuing fixation of the wound margins.

\* The 3M Steri-Strip skin closure tape is available from Minnesota Mining and Manufacturing Co., St. Paul, Minnesota.

#### RESULTS

The results obtained with this method are very good. In 500 patients (960 injections), there were less than 1 per cent failures, notwithstanding the fact that in this period 9 new investigators had to learn the technique. Not only was the number of failures reduced, but the time needed for the surgical-technical part of the procedure up to and including the preparation for the injection was shortened. Before we used the new clamp, needle and forceps, a bilateral injection took from 20 minutes to half an hour; now it takes only 10 to 15 minutes.

#### SUMMARY AND CONCLUSIONS

Of utmost importance in the technique of lymphangiography is the puncture of a vessel. A quick and simple puncture, without the risk of perforation, is the primary requisite for a successful lymphangiographic examination, yet in the literature this point is seldom considered in detail. With the use of the special clamp, needle and forceps here described, the puncture is made much easier and the risk of failure is reduced to a minimum, even when attempted by inexperienced investigators.

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The aforementioned instruments are made and are available in the technical workshop (Head: T. Klein) of the Department of Radiology (Head: Prof. Dr. J. R. von Ronnen) of the University Hospital, Leiden, The Netherlands.





# IMPROVED TECHNIQUE OF LYMPHANGIOGRAPHY\*

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**L**YMPHANGIOGRAPHY is the roentgenologic visualization of the lymphatic system. It has been increasingly accepted as an important adjunct in the diagnosis of and treatment for pathologic conditions involving the lymphatic system. Technical difficulties have remained the greatest obstacle in the performance of lymphangiography. The purpose of the author is to present an improved technique of lymphangiography.

## MATERIALS AND EQUIPMENT

The essential materials and equipment are as follows:

1. Magnifying glasses (Fig. 1A)
2. 20 inch length of P10 polyethylene

catheter with short bevel at the tip (Fig. 1B)

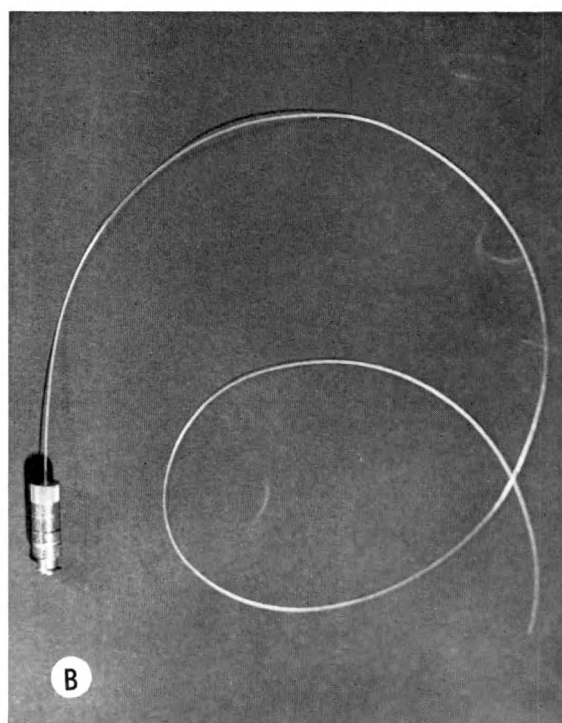
3. Vannas capsulotomy scissors
4. Ethiodol (an ethyl ester of iodized poppy seed oil containing 37 per cent iodine)
5. 0.5 per cent Evans blue dye or 11 per cent patent blue violet (alpha-Zurine 2G)
6. Bulldog clamp (serrofine hemostat) with teeth filed smooth
7. Two per cent xylocaine hydrochloride
8. Gravity injector
9. Cut-down set

## TECHNIQUE PROCEDURE

1. The patient is placed in a supine position on an examining table. The feet are



A



B

FIG. 1. (A) Magnifying glasses. (B) Polyethylene catheter.

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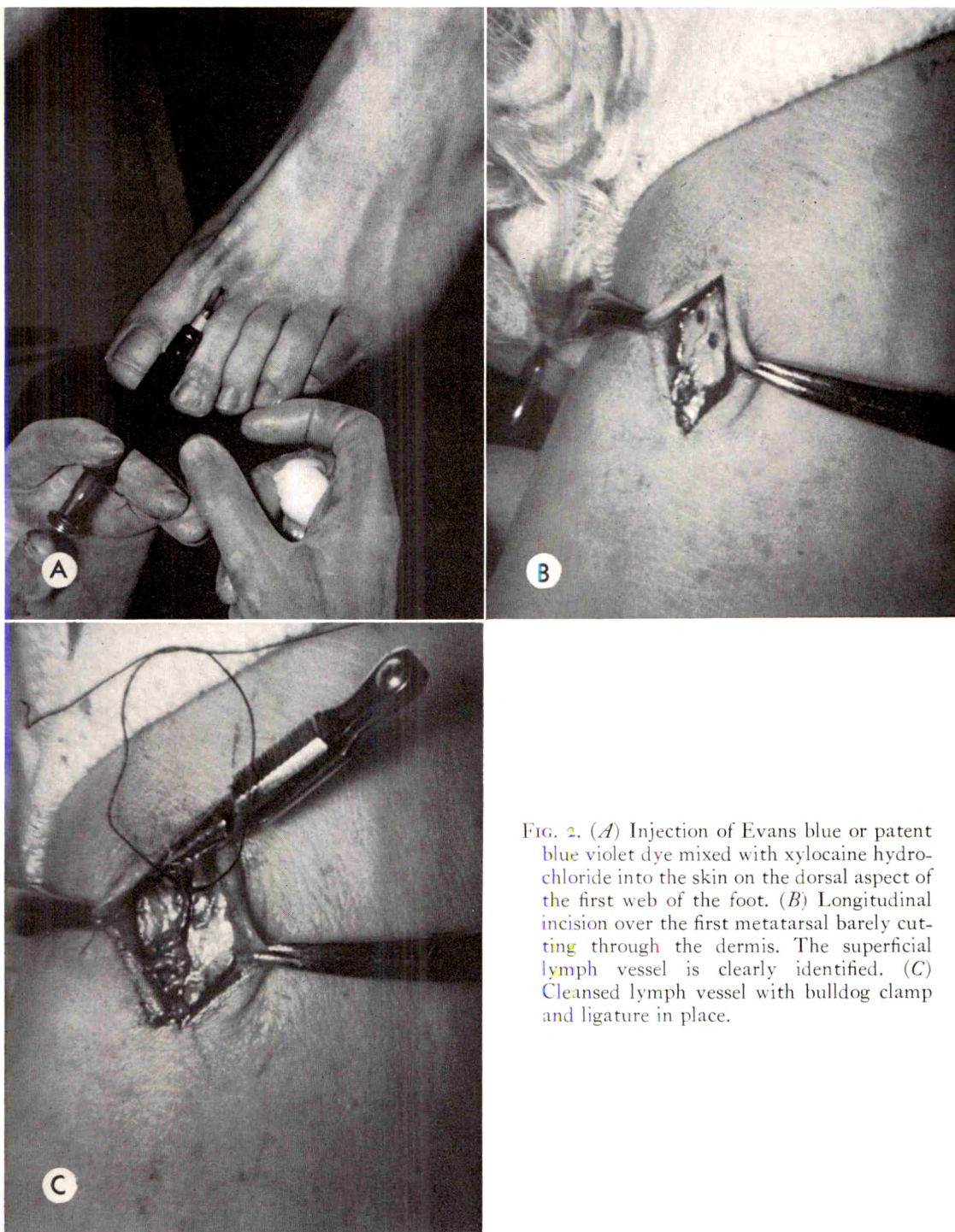


FIG. 2. (A) Injection of Evans blue or patent blue violet dye mixed with xylocaine hydrochloride into the skin on the dorsal aspect of the first web of the foot. (B) Longitudinal incision over the first metatarsal barely cutting through the dermis. The superficial lymph vessel is clearly identified. (C) Cleansed lymph vessel with bulldog clamp and ligature in place.

draped and prepared. One-half ml. of an equal mixture of Evans blue dye and xylocaine hydrochloride or 0.25 ml. of a mixture of equal amounts of patent blue violet and

xylocaine hydrochloride is injected intradermally and subcutaneously on the dorsal aspect of the skin of the first and second webs (Fig. 2A). In upper limb lymph-



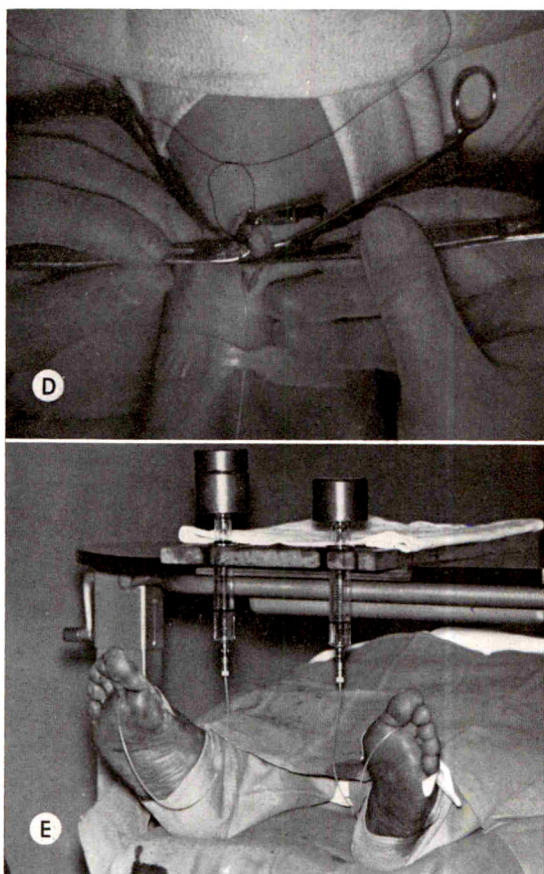


FIG. 2. (D) Insertion of polyethylene catheter through a small hole of the lymph vessel. (E) Injection of ethiodol with gravity injector.

angiography, the dye is injected into the skin on the dorsal aspect of the web of the thumb and second finger.

2. About 5 to 15 minutes later, a local anesthetic (2 per cent xylocaine hydrochloride) is injected into either the first metatarsal or tarsal area. The operator, wearing the magnifying glasses, makes a longitudinal 1 inch skin incision over the first metatarsal, or a transverse incision over the tarsal, barely penetrating the dermis (Fig. 2B). In upper limb lymphangiography, the incision is made either proximal or distal to the anatomic snuffbox. The dye-carrying lymph vessels are identified by the blue streaks.

3. The subcutaneous tissue is explored by blunt dissection. The lymph vessel is identified and occluded proximally by a

bulldog clamp. The lymph vessel can be distended by massaging with the thumb from the web space to the incision. After the vessel is cleaned, a ligature is passed under the lymph vessel (Fig. 2C).

4. A handle of a forcep is placed under the distended lymph vessel over which a small hole is made with Vannas capsulotomy scissors. The polyethylene catheter, which has been immersed in ice water for about 1 hour, is then inserted into the lymph vessel through the hole and is secured with a single tie of the ligature. The catheter is fixed to the foot by a narrow strip of adhesive tape (Fig. 2D).

5. Test injection of a small amount of normal saline will serve to verify an intralymphatic injection, as indicated by flushing the blue dye from the catheter, and from the lymph vessel. The catheter is connected with an adapter to the 20 ml. syringe containing 12 ml. of ethiodol. The syringe is then put vertically into the wooden frame of the gravity injector and a lead cap weighing 9.5 pounds is placed on the plunger (Fig. 2E). The usual injection time is about 2 hours. If there is obstruction in the lymphatic system, especially in the inguinal or pelvic region, the injection time may be longer. In upper limb lymphangiography, 5 to 6 ml. of contrast medium is injected into an upper extremity. Infants and children receive from one-third to two-thirds of the adult dose.

6. After the completion of the injection, the skin incision is sutured and a pressure dressing is applied.

#### ROENTGENOGRAPHIC EXAMINATION

Immediately after the completion of the injection, the patient is transferred to the roentgenographic table. With the patient in a supine position, 14×17 inch anteroposterior roentgenograms of the abdomen and the thorax are made. Oblique and lateral views of the abdomen are always indicated. These roentgenograms afford the best visualization of the lymph vessels (Fig. 3A). A lateral roentgenogram of the thorax is obtained at this time if the thoracic duct



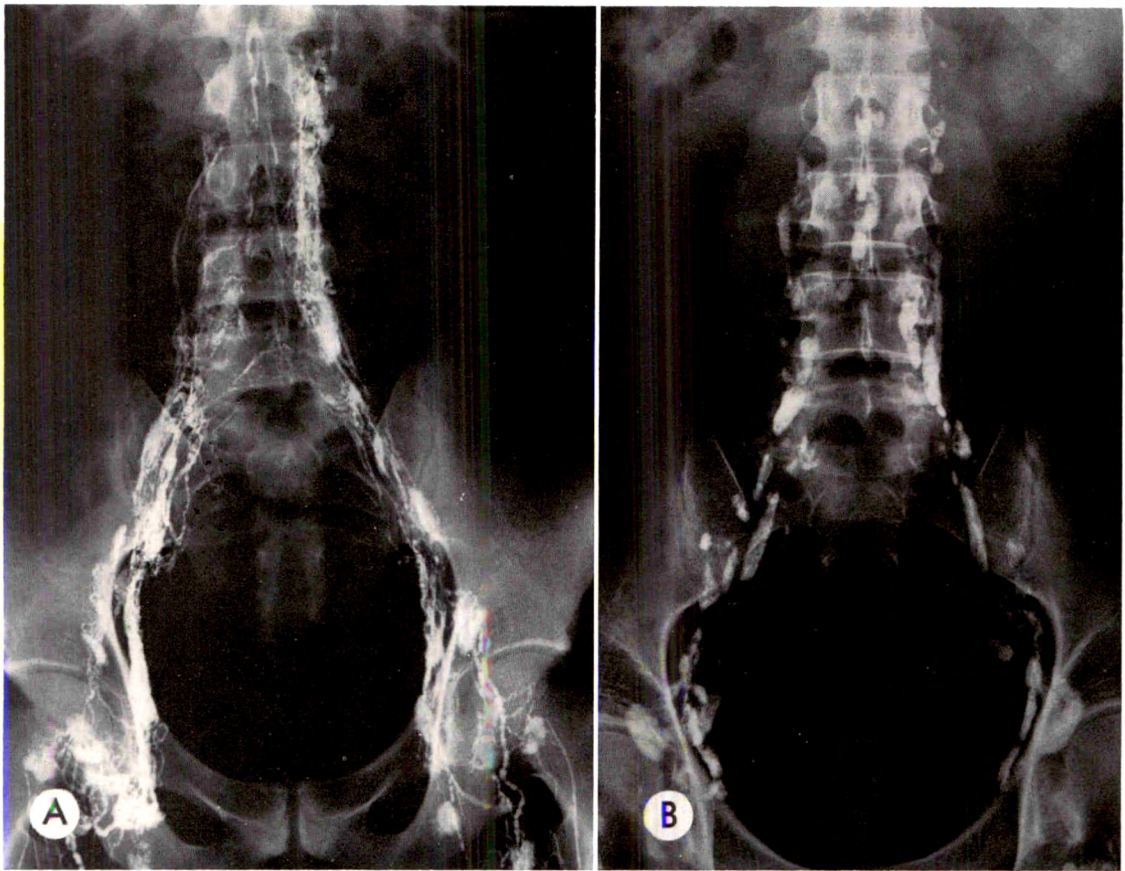


FIG. 3. Normal lymphangiograms. (A) Immediate roentgenogram showing normal appearing lymph vessels and partially filled lymph nodes. (B) Twenty-four hour roentgenogram showing well opacified lymph nodes and emptying of the lymph vessels.

is filled. If the thoracic duct is not visualized on an immediate roentgenogram, anteroposterior and lateral studies of the thorax are obtained in 2 hours. In 24 hours, anteroposterior, oblique and lateral roentgenograms of the abdomen are made. These late roentgenograms show an emptying of the lymph vessels and a good filling of the lymph nodes (Fig. 3B). Sometimes, tomographic studies of the lymph nodes may permit a satisfactory analysis of the position and the architecture of the lymph nodes. The patient may be asked to return for follow-up studies for as long as 12 to 20 months, if the condition warrants.

#### DISCUSSION

Lymphangiography is a specialized examination which requires some skill and

experience. Technically, most of the institutions performing this examination are still using a needle of either 27 or 30 gauge. The failure rate for experienced operators is about 5 to 10 per cent. By using a polyethylene catheter and magnifying glasses, the failure rate in our series has been reduced to less than 1 per cent.

The advantages of the catheter technique are that the tip of the catheter with a short bevel is not as sharp as that of the needle, and that the flexibility of the catheter makes it more adaptable to the curvature of the lymph vessel than does the needle. Therefore, the catheter is less likely to damage the lymph vessel, thereby causing perforation and extravasation of the contrast medium or failure of the examination. Magnifying glasses greatly facilitate



the dissection and cannulation of the lymph vessel.

The difficulties in the performance of lymphangiography are not great and can be overcome. If the incision of the skin is made barely through the dermis and the subcutaneous dissection is carefully performed, undamaged superficial lymph vessels are usually quickly and easily identified. Occasionally, one must explore more deeply into the subcutaneous tissue if the superficial lymph vessel is not revealed. The application of the bulldog clamp needs great care and should be made before the cleaning of the vessel. Careless use of the bulldog clamp can cause laceration of the lymph vessel. This condition may result in leakage and extravasation of contrast medium. For successful cannulation, the lymph vessel should be thoroughly cleaned by a meticulous removal of the surrounding tissues. An uncleaned lymph vessel may lead to perivascular cannulation. In a patient with edema of the extremity, isolation of the lymph vessel may be difficult; however, it can be facilitated by the elevation of the extremity before the examination is started.

#### SUMMARY

1. The technique of lymphangiography is not unduly difficult, though it requires some skill and experience.
2. An improved technique of lymphangiography is described.
3. The advantages of polyethylene catheter and magnifying glasses are presented.
4. Precautions in performing lymphangiography are discussed.

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## SAFETY GUIDESPRING FOR PERCUTANEOUS CARDIOVASCULAR CATHETERIZATION\*

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**S**PRING-GUIDED catheterization has expanded the use and significantly reduced the morbidity of arterial catheterization during the decade since Seldinger<sup>3</sup> and Prioton *et al.*<sup>4</sup> introduced the basic features of the percutaneous technique. Not surprisingly, guidesprings have occasionally broken in use. Usually, the resulting fragments have been left where they came to lie; sometimes surgical or transluminal recovery techniques were used. The complication is not a necessary disadvantage of the technique; it is preventable. It is the primary purpose of the authors to describe a new safety guide wherein spring breakage, if it should occur, will not result in a detached fragment and the possible clinical consequences.

### FIXED VS. MOVABLE CATHETER GUIDE CORES

The Seldinger guide consists of a long, slender coilspring with a fixed wire core stiffening all but the tip. Prioton modified this guide through use of a movable rather than fixed core which, when advanced or withdrawn during the procedure, facilitated retrograde transvalvular catheterization. The writers' personal experiences in literally thousands of guided catheterizations have not justified the routine use of fixed-core guides. On the contrary, in addition to the increased maneuverability of guides with movable, malleable cores, they are—hypothetically at least—less liable to breakage, other things being equal. The reason for this assumption can be found in a review of the various causes of guide breakage.

### CAUSES OF CATHETER GUIDE SPRING BREAKAGE

Coilspring catheter guides are made in one of two ways—either by winding individual springs over a temporary central mandrel on a lathe or by special spring-winding machines which, through the precise application of deforming forces, impart the desired spring configuration. The former has the advantage of allowing the user to make his own springs on a custom basis, thus eliminating the time lag and expense of getting a manufacturer to make up special units. The latter has economic and other important advantages of mass production since the output of the commercial spring-winding machine is a continuous coilspring which can be cut up into segments of any desired length. Standardization is probably more readily maintained. Regardless of how the coilspring is formed, it should be constructed of a suitable stainless steel wire which will undergo repeated flexion without breakage and which will resist permanent deformation under reasonable stress. Even with the best available wire, what is done to it during manufacture and use of the spring will influence the likelihood of guide breakage. What are some of the controllable factors? Chemical alterations in the metal would be likely to occur if it were subjected to electroplating (which is fortunately not done by guide makers). A more important chemical alteration which has surely led to guide breakage can be introduced during manufacture. Spring guides require a finished, smoothly rounded tip in order to avoid scratching or other trauma to the vascular lining and

\* From the University of Oregon's Minthorn Memorial Laboratory for Cardiovascular Research through Radiology. Work aided by grants from the USPHS, H-3275 and H-6336, the Oregon Heart Association, and Mallinckrodt Pharmaceuticals.



wall. This is accomplished by soldering or welding one end of the spring into a solid mass which is ground and buffed to the desired shape and surface. The improper use of acid solder flux is an important possible cause of tip breakage. During the soldering process, flux often migrates one or more inches through the central lumen of the distal spring. If it is not promptly and completely removed, its inevitable effect will be hydrogen embrittlement, an unfavorable alteration in the affected segment of the finished guide. Chemical neutralization or physical removal of flux residue is a difficult and unsure process, especially if ultrasonic techniques are not used. Elevated temperature during the final phase of manufacture has been used to minimize hydrogen embrittlement; it has not solved the breakage problem. A flux or otherwise weakened batch of guide springs can be suspected by fatigue-testing samples, but since this test is *per se* destructive, it cannot be used to exclude the occasional faulty spring. A compromise technique is to fatigue-test random samples from each batch and to give every spring a tug and a twist before inserting it into a patient. In our laboratory this simple routine precaution has detected faulty guides and thereby prevented in-patient breakage. Probably, the most important single cause of spring breakage is not chemical but physical; namely, undue stress during use. Too sharp or too frequent bending of the guide at a given site predisposes to separation. For practical purposes, therefore, it is prudent to replace immediately any spring guide which shows a persistent angular deformity under fluoroscopy and where possible *to use a given guide once only*. The likelihood of repeated same-site flexion at the end of the immovable central core weighs against the indiscriminate use of Seldinger (or other fixed-core) guides for other than the originally intended purpose of percutaneous introduction. In our laboratory, the foregoing policies have been followed for several years. Rarely, has spring breakage occurred within patients. While several

weak springs have been detected and discarded through the use of pre-insertion checks, only 3 of thousands used have broken inside of patients. Two of these involved deliberate extraluminal embedment during the course of transluminal recanalization. In the third, guide breakage should probably not have occurred, since an acute flexion deformity was detected by fluoroscopy, but guide-replacement was delayed until too late. In all 3 instances, recovery efforts were not believed necessary and the patients now bear innocuous reminders of our technical fallibility. To summarize, properly made, tested and utilized springs should not break in use. The fact is, they will; and we are, therefore, compelled to take steps to minimize the ill effects upon our patients. The remainder of this communication relates to the use of inner guard wires designed to prevent separation and therefore permit the prompt removal of broken-off guide tips.

#### A GUARDED, SAFETY GUIDE FOR CATHETERS

The central lumen of a coiled spring guide can accommodate not only the usual movable or fixed core wire but also a second, smaller guard wire which is firmly attached to the tip or both ends of the guide. Guidespring breakage will thus not result in fragment detachment and retrieval is a simple matter. Following our consultation with Mr. William Cook, such a guide was designed and is now commercially available without an increase in cost over the unguarded variety.\* As shown in Figure 1, this type of safety guide (o.d. .047 inch) employs a thin (.008 inch) but strong guard wire attached to the tip of the spring. Also running within the spring is a conventional core wire (.018 inch diameter). It is our preference to leave the .018 inch stiffening wire unattached at either end (to avoid fixed-point stress and to increase control over tip configuration). Distal attachment of stiffening wire to spring some distance

\* Cook Incorporated, 925 South Curry Pike, Bloomington, Indiana.

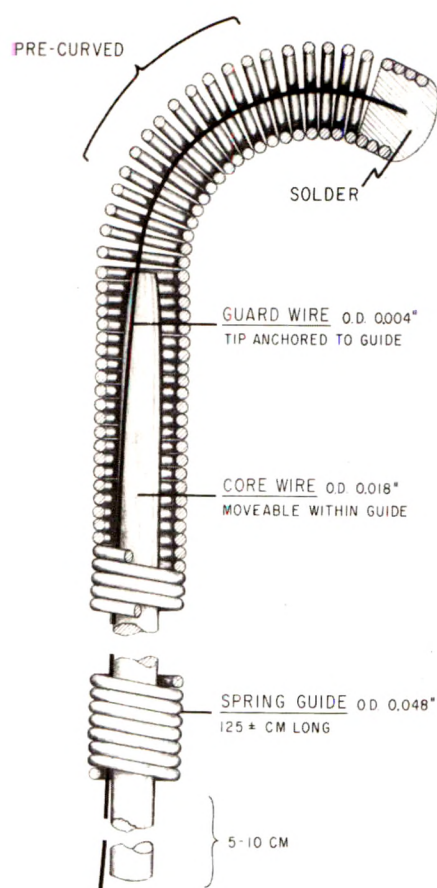


FIG. 1. Diagrammatic sketch of guarded safety catheter guide. Guard wire currently used is .008 inch in diameter, and not shown true scale in sketch.

behind a flexible tip can be achieved if desired. The thin guard wire prevents fragment separation in the event of breakage, but detracts little from tip flexibility. A design advantage is evident in that the guard wire affords a simple method for controlling the configuration of a pre-curved guide tip, a subject meriting brief digression at this juncture.

#### CONTROLLED TIP CONFIGURATION FOR CATHETER GUIDES

If, during the course of a selective catheterization, we are able from the outside to alter the flexibility and configuration of the exploring end of a catheter or its guide, we are better able to pick off desired arterial branches or pass safely through valvular

orifices. It is this basic capability which Prioton added to Seldinger's guide, thereby opening the way to extensive subsequent use of left heart catheterization. Flexibility is easily controlled by advancing or withdrawing the stiffening core. There are a variety of ways of controlling the radius of tip curvature of guide and/or catheter. Perhaps the simplest of these is the use of pre-shaped flexible thermoplastic catheters, pioneered by Ödman.<sup>3</sup> With these and catheters of other materials, a simple guide is usually used for percutaneous insertion and to modify catheter stiffness and configuration during the procedure. There have been developed several techniques for "positive" catheter control. In 1960 Mr. Lowell Edwards designed, made, and provided our laboratory with a prototype catheter that went considerably beyond anything then available through incorporation in its wall of a hollow flexible shaft, braided from 12 strands of fine stainless steel wire. By virtually eliminating shaft torsion, this provided highly accurate torque or rotary control of the catheter. He also designed and constructed a special controlled curvature catheter tip guide. More recently, Viamonte and Parks,<sup>6</sup> and Gensini<sup>1</sup> have reported on similar torque-controlled catheters (marketed by the Cordis Corporation and U. S. Catheter and Instrument Corporation, respectively). In addition to the use of braided wire construction, torque control can be obtained by case hardening ("egg-shelling") the outer surface of certain thermoplastics, a technique used in radiopaque teflon catheters by Dr. Eugene Klatte and Mr. William Cook (personal communications). Yet another means for sophisticated catheter tip control is the special guide system devised by Muller (and marketed by U. S. Catheter and Instrument Corporation).

To the foregoing methods for catheter control, the safety catheter guide reported upon here adds yet another. Through an appropriate combination of deformity and heat, the tip of the safety guide can be formed with a desired curvature. By re-



tracting the guard wire relative to the outer coil spring, the latter can be shortened, compressing the gaps at the periphery of the previously imparted spring curvature. Immediate straightening results. The conventional stiffening wire can, by bending it, also be used to further the variety of tip configurations obtainable (through a combination of its malleability and its mobility within the spring). We have used such guides for several months; none has yet broken.

#### SUMMARY

1. *Guide spring breakage can be lessened* by several means: (1) the proper choice of spring wire, (2) avoidance of chemical weakening (as by acid core solder or electroplating), (3) pre-use testing, (4) in-use avoidance of overflexion, and (5) minimum re-use of spring guides. Attention to the foregoing will minimize but not prevent the breakage of catheter guides.

2. *The clinical importance of guide breakage can be reduced* markedly by the use of a slender, separate guard-wire which, together with the usual fixed or movable central stiffening wire, runs through the center of the guide and which, through attachment to the extreme tip of the guide, prevents separation in the event of spring breakage. The integrity and attachment of this inner guard wire can readily be tested (by attempting to stretch the spring) and its fracture at the time of spring breakage appears to be an extremely unlikely event in the hands of the alert and competent worker. This new safeguarded catheter

guide is commercially available at a cost which permits it to be used as an expendable item.

3. *Guide tip configuration can be externally controlled* by shortening so as to "stack" coils and thereby straighten the axis of a pre-curved guide spring. This approach requires the use of an inner wire attached to the tip of the catheter which would interfere with tip flexibility were the ordinary core wire to be used for this purpose. The safety guide reported here offers means for increasing safety and control without greatly increasing the cost of guided catheterization.

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## PHOTOGRAPHIC SPOT FILMS AND RADIOGRAPHIC SPOT FILMS\*

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**F**LUOROSCOPY allows observation of the dynamic radiographic image and enables the radiologist to expose "spot films" that demonstrate subtle or fleeting findings to their best advantage. Fluoroscopy with conventional equipment can be uneconomical in terms of information obtained compared with the radiation needed. Although no evidence has shown that roentgen rays are harmful at the levels ordinarily used in diagnostic procedures, it is prudent to minimize radiation exposure to all patients, and especially to infants and children. Technical advances during the past decade, including image amplifiers and closed circuit television systems have significantly reduced radiation requirements for fluoroscopy. Since 1960, these electronically enhanced fluoroscopic images have been routinely recorded on television tape<sup>1,2</sup> at this hospital. At least 5 different video-tape recorders are now commercially available for use in radiography.

Video-tape recording of the fluoroscopic image improves consultative services. The referring physician interested in the detailed findings in his patients can review the entire tape recording with or without the fluoroscopist in attendance.

Video-tape recording helps in teaching and training radiologists. The fluoroscopist, often a radiologist in training, records on magnetic tape the entire examination of every patient; because the tape records only the fluoroscopic exposures, the examinations can be reviewed in less time than was required for their actual performance. For example, combined studies that have taken 3 or more hours to perform can be reviewed in less than an hour by the radiological staff as part of the daily activities of their department. In this way,

every resident can have daily fluoroscopic experience without daily exposure to radiation and with economy in time. Working habits of young radiologists can be conveniently and critically evaluated because every examination is reviewed. Finally, through these reviews every patient is examined by experienced members of the departmental staff.

Video-tape playback may not be possible on the hospital wards or in physicians' offices; therefore, other methods are needed to record important fluoroscopic findings. The radiologist can expose spot films conventionally; this requires additional radiation and negates a major advantage of the electronically enhanced magnetically recorded image. As much as 5 minutes of the fluoroscopic examination can be recorded on magnetic tape with the amount of radiation needed for one single film exposure. "Photographic spot films" made from the tape recorded image during playback of the tape require no additional radiation and afford permanent records of the examination.

### TECHNICAL DETAILS

The basic equipment necessary to make photographic spot films consists of a tripod mounted 35 mm. camera placed in front of a television monitor (Fig. 1). The camera is equipped with an iris type (nonfocal plane) shutter, an F2 lens, and is set at a shutter speed of 1/30th second. This speed records a fully interlaced television image. High speed films such as Kodak Tri X or DuPont 928B are used. A darkened viewing room eliminates ambient light.

The exposed film is simply and rapidly developed in any automatic x-ray film processor, which also effectively increases

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FIG. 1. The basic equipment for photographic spot films of the tape recorded fluoroscopic image consists of a 35 mm. camera mounted on a tripod placed in front of a television monitor.

the speed of the film threefold and enhances its contrast accordingly. The photographic spot films are viewed or projected on standard film strip equipment (Fig. 2).

The use of two television monitors, one

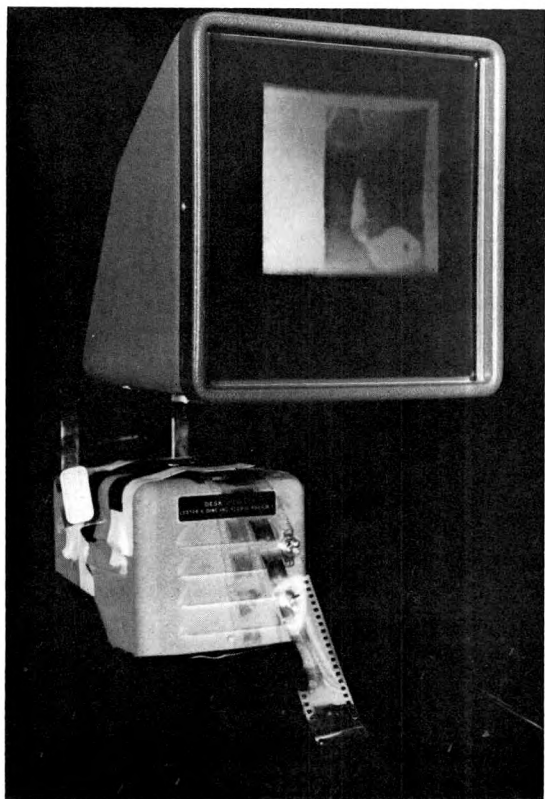


FIG. 2. The photographic spot film strip is viewed on any standard film strip equipment.

specifically for viewing, and the second equipped with a blue phosphor kinescopic tube for photographic recording with blue sensitive film (DuPont 834B), permits simultaneous unobstructed viewing and photographic recording without the necessity of absolute darkness in the viewing room (Fig. 3). A rapid film advance accessory and a 200 feet film magazine allow uninterrupted photographic spot filming. Figure 4, *A* and *B* shows close-up views of the camera and accessories, mounted on top of a commercially available Kine recorder.

#### DISCUSSION

Advantages of photographic spot films over conventional radiographic spot films include:

1. *No added radiation to the patient.* Many photographic spot films can be obtained in rapid succession (up to 6 per second) without any additional radiation to the patient, since the image on the photographic film is formed by light rays from the monitoring screen and *not* by roentgen rays passing through the patient (Fig. 5).

2. *Insight by hindsight.* Often the radiologist sees the important findings to better advantage after he has exposed a significant number of radiographic spot films. He must then decide whether further spot films with added radiation are warranted for a more convincing demonstration of important

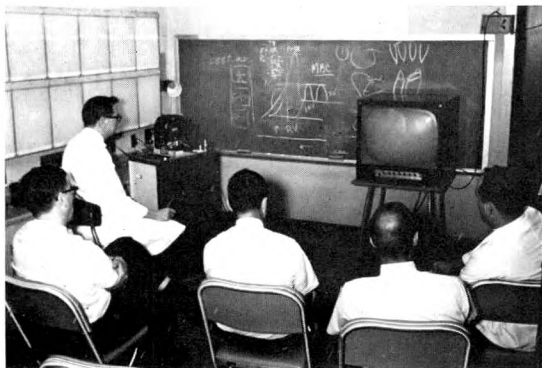


FIG. 3. The use of two television monitors enables simultaneous unobstructed group viewing and recording of the fluoroscopic image.

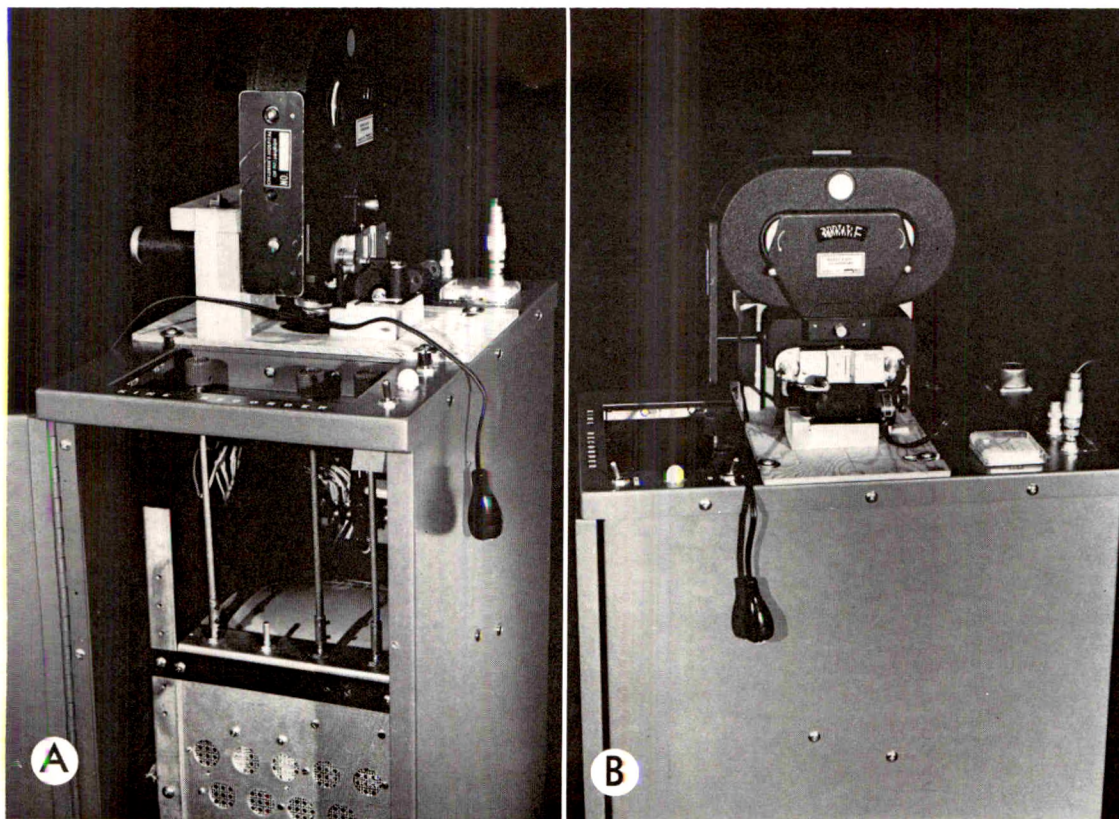


FIG. 4. (A and B) Close-ups of the camera equipped with a 200 foot film magazine and a rapid film advance mechanism mounted on a commercially available kine-recorder. This monitor is equipped with a blue phosphor and blue sensitive photographic film is used. The review and record session can be carried out without the necessity of total darkness in the viewing room.

findings. This problem is eliminated with photographic spot films. After review of an examination recorded on tape, the best observations can be chosen for the permanent record on photographic spot films. In effect, these represent "retrospective spot films."

3. *No time lag between observation and recording.* Conventional spot films cannot be immediately exposed during the fluoroscopic examination because of the necessary delay involved in bringing the roentgenographic cassette into position. The radiologist exposing spot films hopes to show what he saw on direct fluoroscopy. All too often, this spot film exposure does not corroborate the fluoroscopic observation. Continuous cine radiographic studies<sup>3</sup> eliminate this problem, but require relatively

high radiation levels and special cine radiographic equipment. The photographic spot film recorded from the television playback allows choice of proper image and eliminates the time lag between the fluoroscopic observation and the film exposure. The radiologist reviews the tape with the certainty that he is photographing the pertinent parts of the examination.

4. *Uninterrupted observations.* In conventional fluoroscopy, observation is interrupted every time a radiographic spot film is obtained. Tape review eliminates such interruption.

5. *Low film costs.* With 35 mm. photographic spot films, the cost of each image is a small fraction of that of conventional radiographic spot films. This does not suggest economic advantages of the method,



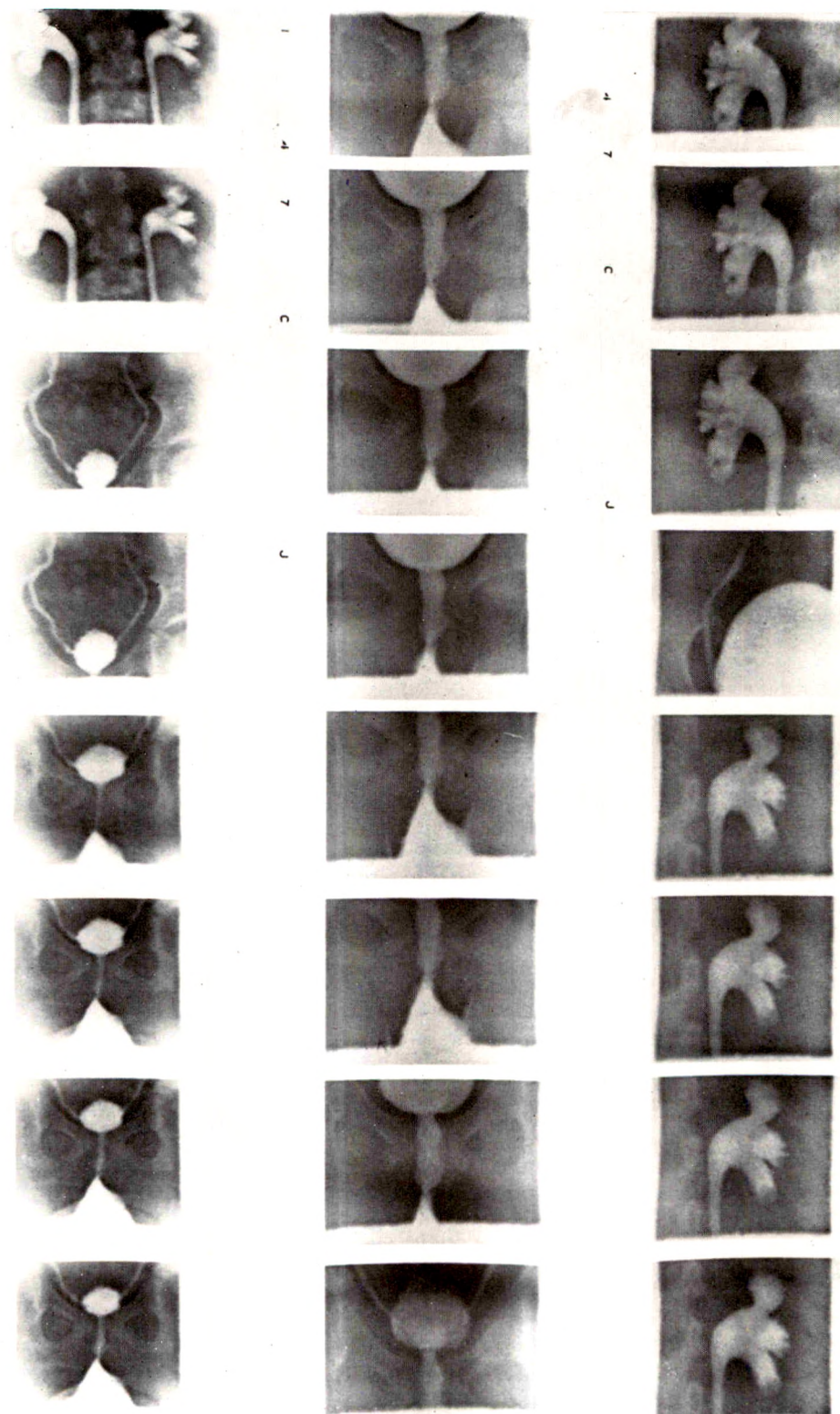


FIG. 5. Direct positive contact prints of the photographic spot film from a tape recorded cystogram illustrate the quality of these images.

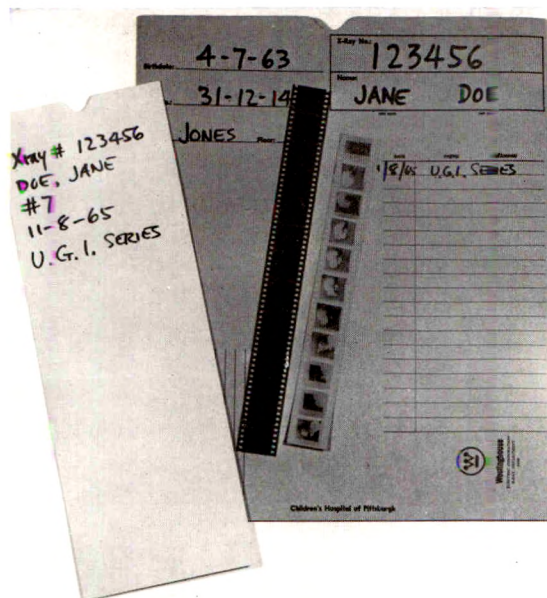


FIG. 6. The individual film strip (left) is kept in a special envelope which is filed with the standard roentgenograms. Copies (right) of the film strip can be made on a Kalvaray film duplicator. These copies can be sent to the referring physician for his record, or placed in the patient's chart.

but the lower cost of the photographic spot films helps cover the additional cost of the electronic equipment.

#### SUMMARY

Fluoroscopic examinations can be recorded on television tape at low radiation

levels making possible better teaching and training of young radiologists and better service to referring physicians.

Important findings can be recorded photographically from the image on the monitoring screen during a subsequent playback of the recorded tape (photographic spot films), at a small cost and with no added radiation to the patient. Photographic spot films on 35 mm. film can be processed in regular automatic x-ray processors, with no special equipment or personnel. These filmstrips are filed in special envelopes within the regular radiographic folder of each patient (Fig. 6).

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## INVESTIGATIONS IN COLORED RADIOGRAPHY\*

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MAN lives today surrounded by a world of color. On all sides, he sees the myriad of hues produced by both nature and his fellow man. Struck by the sheer brilliance and beauty that color gives to his world, he is, nevertheless, aware that the three dimensional quality of color provides another function of major importance: the imparting of information.

Only a few examples need be mentioned of the additional capacity for information transmittal contained in colored media as compared to black and white. The impact of colored motion pictures, color television, and the use of color in advertising is self-explanatory. The person with normal color vision has tremendous advantages in everyday living over the color-blind or color-deficient person. In the fields of photogrammetry and aerial photography, colored pictures have proven to have very definite advantages over black and white pictures. The underlying reason for these advantages is the basic fact that black and white images are limited to one parameter; brightness, whereas colored images have three parameters; brightness, hue and saturation. If the three parameters of color are thought of as axes on a three dimensional graph, color can be thought of in terms of a "color space." Black and white images are limited to one dimension in color space. The additional parameters of color permit more information to be conveyed.

There are no sharp boundaries between the various regions of the electromagnetic spectrum, and properties of one region may be common to another region also.

The similarities between light and x-rays are discussed in an article by Tasker.<sup>30</sup> Both types of radiation can be produced and controlled by electrical equipment, and both can be polarized, refracted, absorbed and scattered as well as made to cause fluorescence. Both types also can interact with a photographic emulsion to produce an image.

Recognizing the advantages of colored images, a number of investigators has attempted to produce colored radiographs.<sup>2-10,14,16,20,25,27,29</sup> Donovan, the first investigator, performed a variation of Maxwell's experiment in colored photography.<sup>8,9,10</sup> He made a series of three radiographs of a subject, taken at three different energies, and treated them as separation negatives. He superimposed the images of these three radiographs on a white screen while projecting them through red, green, and blue filters. A colored image resulted.

Most of the past attempts at attaining colored radiographs have been empirical. The scientific processes involved have not been delineated. The purpose of the authors is to discuss the various factors involved in the radiographic process, the interrelation of these factors, and how the process can best be altered to obtain more information about the subject.

### THE RADIOGRAPHIC PROCESS

The three essential components in radiography are the x-ray source, the subject and the recording medium (in this case a photographic emulsion). The subject is between the source and the recording medium

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From the Department of Environmental Engineering, <sup>†</sup> the X-Ray Laboratory of the Department of Physics, <sup>‡</sup> and the Color Measurement Laboratory of the Department of Chemistry, Rensselaer Polytechnic Institute, <sup>§</sup> Troy, New York.

This paper is based on part of a thesis submitted by Harvey R. Prins to the Graduate School of the Rensselaer Polytechnic Institute in partial fulfillment of the requirements for the degree of Doctor of Engineering in the Department of Environmental Engineering.

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and the resulting image is a "shadow-graph." The information that can be obtained from the resulting image is a function of these three major components. Normally, the subject is unknown but for the purposes of this study it was well defined quantitatively and qualitatively.

Mathematically, the radiographic process can be described by the relationship:

$$D = \int I(E)T(E)R(E)dE$$

where

$E$  is the energy of the x-ray photon,  
 $D$  is the density on the processed radiograph,

$I(E)$  is the intensity-distribution function of the x-ray source,

$T(E)$  is the spectral-transmission function of the subject, and

$R(E)$  is the spectral-response function of the detector.

The density is also influenced by the time of processing and the temperature and concentration of the reagents in processing. However, these can be controlled to maintain constant conditions.

It should be noted the  $I(E)$  is in terms of photons per energy increment. For direct exposure to x-rays, the density is linearly proportional to exposure over a considerable region.

Unfortunately, the above function cannot be expressed in analytical form. However,  $I(E)$  can be specified by a spectral energy-distribution curve,  $T(E)$  by a spectral-transmission curve, and  $R(E)$  by a spectral-response curve. When these three curves are multiplied together, ordinate by ordinate, a spectral-stimulus curve results. The area under the spectral-stimulus curve is directly proportional to the density on the finished radiograph.

In this study, the three curves mentioned above were determined from data available in the literature and the area under the resulting spectral-stimulus curve was compared to the measured densities of the processed radiographs.

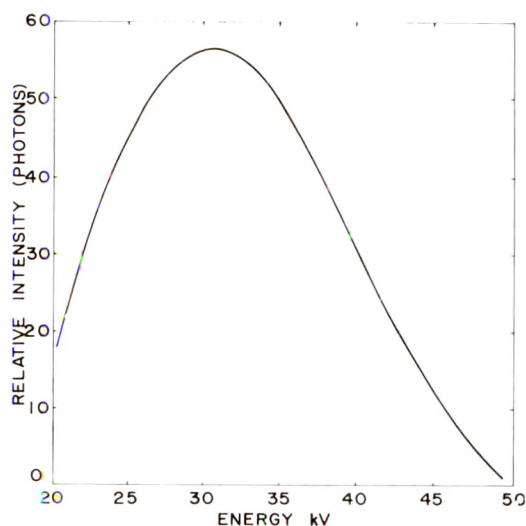


FIG. 1. Spectral distribution curve.

#### X-RAY SOURCE

The x-ray source for this study was a General Electric model R-2 Diagnostic X-ray Unit. This unit has a self-rectified circuit and uses a tungsten target Coolidge tube. Throughout the course of this study, the unit was operated at 50 KVP (kilovolts peak) with a focal spot of 2 square millimeters. One millimeter of Al filtration was permanently fixed at the tube port.

Only a small portion of the center of the

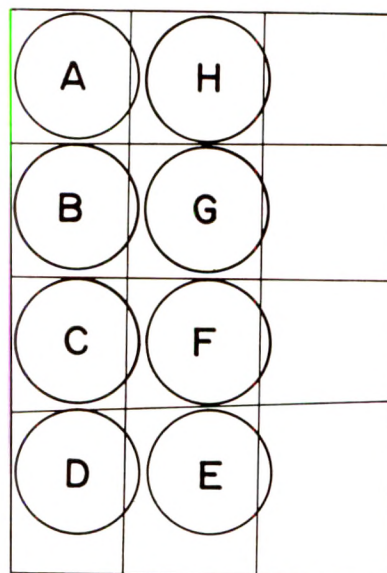


FIG. 2. Plan view of the subject.



TABLE I  
COMPONENTS OF THE SUBJECT

Component	Material	Thickness
Background	none	none
A	thick bone	1350 mg./cm. <sup>2</sup>
B	thin bone	358 mg./cm. <sup>2</sup>
C	medium bone	730 mg./cm. <sup>2</sup>
D	thin chloramine T	598 mg./cm. <sup>2</sup>
E	thick chloramine T	1337 mg./cm. <sup>2</sup>
F	thin dimethylglyoxime	599 mg./cm. <sup>2</sup>
G	medium dimethylglyoxime	1210 mg./cm. <sup>2</sup>
H	thick dimethylglyoxime	1950 mg./cm. <sup>2</sup>

field was used in order to minimize the "heel effect." A picture of the anode taken by the pinhole camera technique revealed an imperfection on the target. However, measurements of exposed film made with a microphotometer showed the field to be uniform.

The spectral distribution curve (Fig. 1) for the x-ray source was obtained from a report by Aitken and Dixon.<sup>1</sup> These authors were the only ones who gave data for self-rectified units. They determined the spectral distribution curve by scintillation spectrometry using a NaI (Tl) crystal and a multichannel analyzer. They explain in detail how the data were corrected for the resolution of the detector and for fluorescence losses from iodine.

#### SUBJECT

A diagram of the subject is shown in Figure 2. The components, listed in Table I, were chosen with some regard to subjects that might be encountered in medical radiography, although no intensive study was made of the composition and density of tissue. The components of the subject were made of reagent grade chemicals pressed into pellet form with a hydraulic press. This assured uniform density as confirmed by the radiographs.

The components called "bone" were made of a known mixture of  $P_2O_5$  and  $Ca(OH)_2$ . This mixture approximates the major constituents of bone.<sup>19</sup> The crystal structure and chemical composition of bone

can be more accurately approximated by the hydroxyapatite,  $3Ca_3(PO_4)_2 \cdot Ca(OH)_2$ ; however, this mineral was not available in the desired form.

The subject component chloramine T,  $1-CH_3C_6H_4-4-SO_2NCINa \cdot 3H_2O$ , was taken to represent some of the heavier organic substances present in tissue.

It should be pointed out that no great pains were taken to represent a medical subject, the essential factor being that the subjects could be described quantitatively and qualitatively.

The subject components were weighed on a precision analytical balance and the thickness of each was determined in milligrams per square centimeter. The components were then hermetically sealed in a box with 5 mil thick mylar windows. A grid was constructed on the bottom of the box with 0.01 inch stainless steel wire. This grid facilitated measurements made with the microphotometer and also aided in getting radiographs in register in a later part of the experiment.

The background of the subject is the unattenuated beam.

The spectral transmission curve for the subject is shown in Figure 3. The data to compute this curve were taken from a report by Stainer.<sup>28</sup> In this report the data given by S. J. M. Allen were used.

Since only an approximation of the radio-

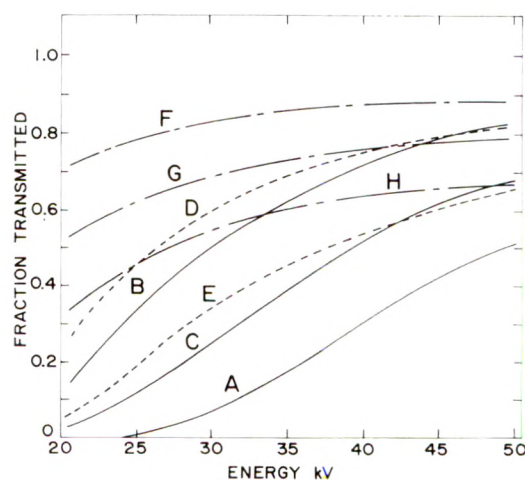


FIG. 3. Spectral transmission curve.

and the resulting image is a “shadow-graph.” The information that can be obtained from the resulting image is a function of these three major components. Normally, the subject is unknown but for the purposes of this study it was well defined quantitatively and qualitatively.

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The density is also influenced by the time of processing and the temperature and concentration of the reagents in processing. However, these can be controlled to maintain constant conditions.

It should be noted the  $I(E)$  is in terms of photons per energy increment. For direct exposure to x-rays, the density is linearly proportional to exposure over a considerable region.

Unfortunately, the above function cannot be expressed in analytical form. However,  $I(E)$  can be specified by a spectral energy-distribution curve,  $T(E)$  by a spectral-transmission curve, and  $R(E)$  by a spectral-response curve. When these three curves are multiplied together, ordinate by ordinate, a spectral-stimulus curve results. The area under the spectral-stimulus curve is directly proportional to the density on the finished radiograph.

In this study, the three curves mentioned above were determined from data available in the literature and the area under the resulting spectral-stimulus curve was compared to the measured densities of the processed radiographs.

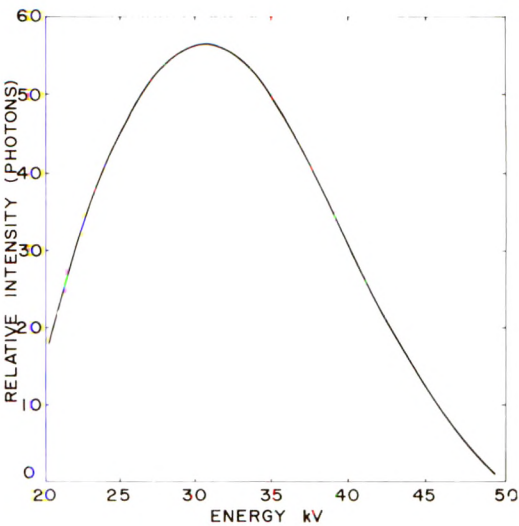


FIG. 1. Spectral distribution curve.

X-RAY SOURCE

The x-ray source for this study was a General Electric model R-2 Diagnostic X-ray Unit. This unit has a self-rectified circuit and uses a tungsten target Coolidge tube. Throughout the course of this study, the unit was operated at 50 KVP (kilovolts peak) with a focal spot of 2 square millimeters. One millimeter of Al filtration was permanently fixed at the tube port.

Only a small portion of the center of the

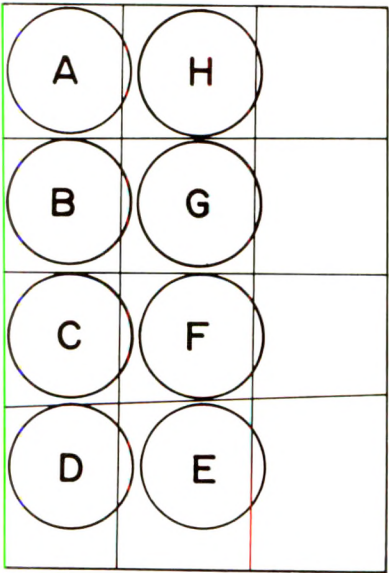


FIG. 2. Plan view of the subject.



TABLE I  
COMPONENTS OF THE SUBJECT

Component	Material	Thickness
Background	none	none
A	thick bone	1,350 mg./cm. <sup>2</sup>
B	thin bone	358 mg./cm. <sup>2</sup>
C	medium bone	730 mg./cm. <sup>2</sup>
D	thin chloramine T	598 mg./cm. <sup>2</sup>
E	thick chloramine T	1,337 mg./cm. <sup>2</sup>
F	thin dimethylglyoxime	599 mg./cm. <sup>2</sup>
G	medium dimethylglyoxime	1,210 mg./cm. <sup>2</sup>
H	thick dimethylglyoxime	1,950 mg./cm. <sup>2</sup>

field was used in order to minimize the "heel effect." A picture of the anode taken by the pinhole camera technique revealed an imperfection on the target. However, measurements of exposed film made with a microphotometer showed the field to be uniform.

The spectral distribution curve (Fig. 1) for the x-ray source was obtained from a report by Aitken and Dixon.<sup>1</sup> These authors were the only ones who gave data for self-rectified units. They determined the spectral distribution curve by scintillation spectrometry using a NaI (Tl) crystal and a multichannel analyzer. They explain in detail how the data were corrected for the resolution of the detector and for fluorescence losses from iodine.

#### SUBJECT

A diagram of the subject is shown in Figure 2. The components, listed in Table I, were chosen with some regard to subjects that might be encountered in medical radiography, although no intensive study was made of the composition and density of tissue. The components of the subject were made of reagent grade chemicals pressed into pellet form with a hydraulic press. This assured uniform density as confirmed by the radiographs.

The components called "bone" were made of a known mixture of  $P_2O_5$  and  $Ca(OH)_2$ . This mixture approximates the major constituents of bone.<sup>19</sup> The crystal structure and chemical composition of bone

can be more accurately approximated by the hydroxyapatite,  $3Ca_3(PO_4)_2 \cdot Ca(OH)_2$ ; however, this mineral was not available in the desired form.

The subject component chloramine T,  $1-CH_3C_6H_4-4-SO_2NCINa \cdot 3H_2O$ , was taken to represent some of the heavier organic substances present in tissue.

It should be pointed out that no great pains were taken to represent a medical subject, the essential factor being that the subjects could be described quantitatively and qualitatively.

The subject components were weighed on a precision analytical balance and the thickness of each was determined in milligrams per square centimeter. The components were then hermetically sealed in a box with 5 mil thick mylar windows. A grid was constructed on the bottom of the box with 0.01 inch stainless steel wire. This grid facilitated measurements made with the microphotometer and also aided in getting radiographs in register in a later part of the experiment.

The background of the subject is the unattenuated beam.

The spectral transmission curve for the subject is shown in Figure 3. The data to compute this curve were taken from a report by Stainer.<sup>28</sup> In this report the data given by S. J. M. Allen were used.

Since only an approximation of the radio-

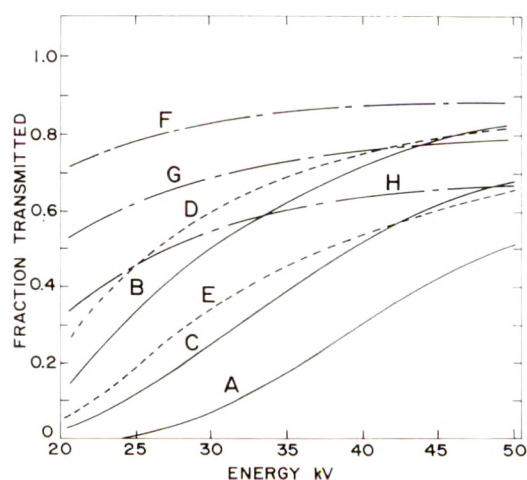


FIG. 3. Spectral transmission curve.

graphic process was sought, it was justifiable to use absorption coefficients determined in a narrow beam attenuation for broad beam application. The attenuation process in the energy region under consideration occurs predominantly by the photoelectric effect. There is considerable coherent scattering at lower energies but these photons have been removed from the beam by the 1 millimeter aluminum filter. The fluorescence yields for the elements in the subject are low and their characteristic emission x-rays are of low energy, so this factor does not interfere.

Unfortunately, facilities were not available to measure the spectral transmission curve directly.

#### RECORDING MEDIUM

The recording medium chosen for this study was Kodak Non-Screen Medical X-ray Film. The general characteristics of this particular film have been investigated in more detail than those of most other films. Among the reasons for the choice are the following:

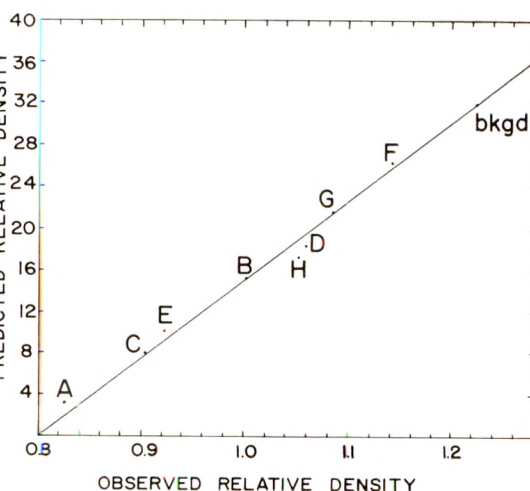


FIG. 5. Observed vs. predicted relative density.

1. The reciprocity law holds for a direct exposure to x-rays.<sup>11</sup>
2. Density is linearly proportional to exposure over a wide density range.<sup>11</sup>
3. The parameters of processing are well understood and can easily be controlled.<sup>12</sup>
4. The spectral response to x-rays has been investigated.<sup>26</sup>

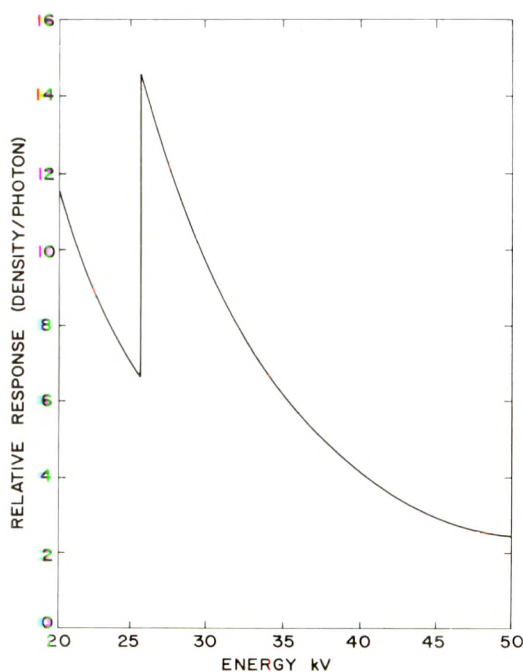


FIG. 4. Spectral response curve.

The spectral response curve for the x-ray film is shown in Figure 4. This curve was plotted from data determined by Seeman.<sup>26</sup>

The 3 curves mentioned previously were multiplied together, ordinate by ordinate, to obtain the spectral stimulus curve for each component. The areas under these curves were determined and called "predicted relative density." These values are compared with "observed relative density" of the processed radiographs. The results are shown in Figure 5.

The observed relative densities were obtained from readings taken on an Hilger L-470 microphotometer.

A line was fitted to the points shown in Figure 5 by the method of least squares. The correlation coefficient was 0.974.

This means that the radiographic process can be quantitatively described by describing the spectral-distribution curve, spectral-transmittance curve and spectral-response curve for the source, object and



recording medium used. However, the data in the literature leading to these curves cover only a limited energy range.

#### COLORED RADIOGRAPHY—COLORIMETRY

The basic principles of the specification of color are given elsewhere.<sup>15,18</sup> However, a brief review may be helpful to the understanding of some of the experimental and computational work.

One basis for the specification of color is the experimental fact that any given color can be matched by a mixture of known amounts of three other colors, known as primary colors. Experimentally, it may be necessary to add one of the primary colors to the unknown and then match this mixture with a combination of the other two known colors.

The amounts of each of the primary colors required to produce a match are called "tristimulus values." If one of the primaries has to be added to the unknown to obtain match, then the tristimulus value for that primary is given a negative sign. Otherwise, the tristimulus values are positive.

Any three primaries may be used provided that no mixture of two of them would match the third. A common choice for the primaries is red, blue, and green.

If one set of primaries is used to measure a second set of primaries, data taken by the first set can be transformed into data of the second set by a homogeneous linear transformation.

In 1931 the Commission Internationale de l'Eclairage (CIE) recommended the adoption of three primaries which are, in fact, not physically realizable. The use of these primaries keeps all the tristimulus values positive. These primaries are defined in terms of the amounts of each of them which must be added together to obtain the spectrum colors. These amounts  $\bar{x}, \bar{y}, \bar{z}$  are the tristimulus values for the spectrum colors based on the CIE primaries. They represent in numerical terms the color matching capability of the normal human eye.

Since  $\bar{x}, \bar{y}, \bar{z}$  are defined for unit irradiance, it is necessary to multiply them by the spectral distribution function,  $E$ , where  $E$  defines a source-object spectral-energy distribution, and integrate the products' over all wavelengths. Thus, in general the tristimulus values are:

$$X = \int E \bar{x} d\lambda$$

$$Y = \int E \bar{y} d\lambda$$

$$Z = \int E \bar{z} d\lambda$$

where  $E, \bar{x}, \bar{y}, \bar{z}$  are all functions of wavelength. Because of the choice of the CIE primaries,  $\bar{x}, \bar{y}, \bar{z}$  are not expressible in explicit form. In addition, it is usually true that  $E$  is not expressible in any simple closed form. For this reason, it is necessary to divide the spectrum into segments and use summations which approximate the previous equations.

It is generally more convenient to use nondimensional quantities, so the chromaticity coordinates  $x, y$ , and  $z$  are introduced.

They are defined by the equations:

$$x = \frac{X}{X + Y + Z}$$

$$y = \frac{Y}{X + Y + Z}$$

$$z = \frac{Z}{X + Y + Z}$$

Only two of the three chromaticity coordinates  $x, y$ , and  $z$  are independent since from the definitions it is evident that

$$x + y + z = 1.$$

Any two of the three chromaticity coordinates,  $x, y$ , and  $z$ , may be plotted in two dimensions. Such a plot, usually with  $x$  as the abscissa and  $y$  as the ordinate, is known as a chromaticity diagram. It is customary to plot on the chromaticity

diagram the points which correspond to the spectrum colors. Such a diagram is given in Figure 6.

The relative luminosity function is the sensitivity of an average normal observer to radiant energy of various wavelengths as far as the judgment of lightness is concerned. The luminous flux is the time rate of flow of luminous energy.

With considerable foresight, the CIE chose  $\bar{y}$  to be the relative luminosity curve. This means that the tristimulus value  $Y$  is directly proportional to the luminous flux in the light being considered. The tristimulus value  $Y$  is the lightness of a color.

Since color has three parameters, it is necessary to give three quantities to specify a color. The two chromaticity coordinates,  $x$  and  $y$ , and the tristimulus value  $Y$  form one set of the three quantities that can be used. Another set is to give the amounts (tristimulus values) of any three primaries which are required for a match. Still another method is to specify the dominant wavelength, the purity and the lightness of a color.

The dominant wavelength and purity can be found from the chromaticity diagram in the following manner. The neutral point is located at chromaticity coordinates of the light source used for viewing. For CIE Source C, an approximation representing average daylight, this point is  $x=0.3101$ ,  $y=0.3165$ . A line can be drawn from the neutral point through the point on the diagram which represents the given color. When this line is extended, it will intersect the spectrum locus. The wavelength at which the intersection occurs is the dominant wavelength for the color.

The straight line on the chromaticity diagram between 400 nm\* and 700 nm represents the purples (mixtures of blue and red), which are not spectrum colors. If a line drawn from the "neutral point" through the sample point intersects the line from 400 nm to 700 nm, then the dominant wavelength for that sample color

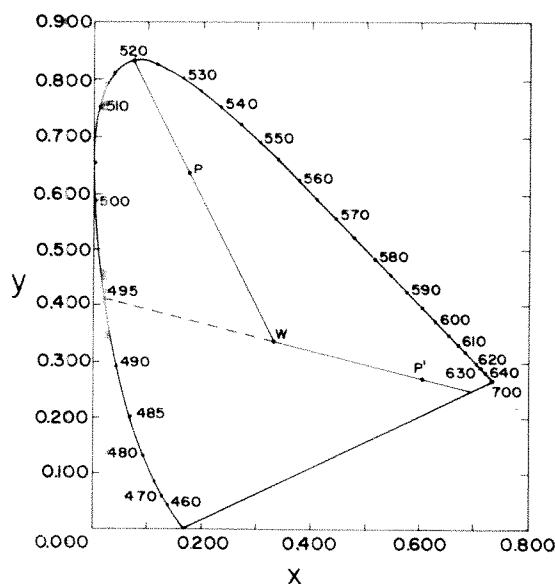


FIG. 6. Chromaticity diagram.

is labeled as the dominant wavelength of its complementary color. This is the wavelength of the point of intersection of the line through the neutral point and the sample point extrapolated to intersect the spectrum locus.

Purity is the ratio of the distance between the sample point and the neutral point to the distance between the spectrum locus and the neutral point.

The dominant wavelength and purity for sample point  $P$  on the chromaticity diagram of Figure 6 are 520 nm and 60 per cent, respectively. The dominant wavelength and the purity for the sample point  $P'$  are 495 nm (complementary) and 75 per cent, respectively.

The foregoing discussion was presented as a brief review of the science of colorimetry. More complete discussions may be found in many articles in the literature.<sup>15,18,21</sup>

#### OPTIMUM COLOR PROCEDURE

Radiographers have long known that the appearance of a radiograph depends on the voltage applied to the x-ray tube, just as photographers know that the appearance of a photograph depends on the color of the light incident on the subject.

\* nm = nanometer, the currently recommended name for millimicron. One (1) nm = ten (10) Å.



TABLE II  
RELATIVE TRANSMITTANCE OF SUBJECT COMPONENTS

Component	$I_0/I_t$		
	25 KV	35 KV	45 KV
Background	1.000	1.000	1.000
A	0.952	0.676	0.380
B	0.457	0.234	0.162
C	0.775	0.408	0.240
D	0.347	0.200	0.158
E	0.662	0.355	0.246
F	0.162	0.138	0.132
G	0.246	0.186	0.170
H	0.339	0.246	0.219

Examination of the spectral transmission curve (Fig. 3) in the previous part shows that the magnitude of transparency and even the order of transparency of the subject components varies at different energies. It can also be seen, from an examination of the spectral transmission curve, why radiographs taken at low energies show much more contrast than those taken at high energies; in the low energy regions, the ordinates for the various components are much further apart than at high energies. If three radiographs are taken with different mono-energetic x-rays, they can be treated like separation negatives in colored photography. The introduction of arbitrary color in the recombination will permit more information to be obtained than is the case in conventional radiography.

For purposes of illustration, an example is calculated for the case when the mono-energetic x-rays are chosen at 25 KV, 35 KV, and 45 KV. If the subject is exposed to x-rays of these energies, the densities of the components will be directly proportional to the ordinates of the spectral transmission curve at these points.

The densities are converted over to fractional transmission by the basic relationship:

$$D = \log I_0/I_t$$

These are tabulated in Table II.

If the radiographs obtained in the above

manner were illuminated by spectrally pure lights obtained by taking narrow wavelength regions from a dispersed beam and their images projected in register on a white screen, the widest physically realizable spread of colors would result.

Since color is not being reproduced but being introduced arbitrarily, a large number of combinations is possible. However, since the eye is more sensitive to changes in hue than in purity or brightness, the recombination should be such that the chromaticity coordinates of the components fall around the neutral point.

For purposes of illustration, the combination shown in Table III will be considered.

The amounts were so chosen that the components would fall around the neutral point.

The chromaticity coordinates for the red light are  $x=.7347$ ,  $y=.2653$ ; for the green light,  $x=.0743$ ,  $y=.8338$ ; and for the blue light,  $x=.1733$ ,  $y=.0048$ .

Using the foregoing data and the laws of color mixing, the chromaticity coordinates for the various components were computed. These are tabulated in Table IV and plotted in Figure 7.

The dominant wave length and per cent purity tabulated in Table IV were obtained from a large chromaticity chart found in Hardy's "Handbook of Colorimetry."<sup>17</sup>

The relative lightness tabulated in Table IV is the weighted sum of the fractional transmission of the 3 radiographs.

The color designation of the subject components was changed from the CIE system to the Munsell system and the

TABLE III  
ILLUMINATING LIGHTS

Radiograph Voltage	Illuminating Color	Wavelength	Amount
25 KV	red	700 nm	0.421
35 KV	green	520 nm	0.631
45 KV	blue	400 nm	1.000

nm = nanometer; 1 nm = 10 Å.

TABLE IV  
COLORS OF THE SUBJECT COMPONENTS

Component	CIE Coordinates		Dominant Wavelength	Per Cent Purity	Relative Lightness*	Munsell Designation		ISCC-NBS Color Name
	x	y						
Background	0.2580	0.3132	490	20	0.17	8 BG	5/4	Moderate bluish green
A	0.3247	0.3841	560	22	1.00	7.5 GY	9/4	Light yellow green
B	0.3593	0.3484	584	22	0.42	7.5 YR	7/3	Light yellowish brown
C	0.3647	0.3671	579	28	0.68	1 Y	8.5/4	Pale yellow
D	0.3349	0.3364	580	12	0.37	10 YR	6.4/1	Yellowish gray
E	0.3527	0.3498	581	20	0.62	9 YR	8/3	Pale orange yellow
F	0.2766	0.3179	492	13	0.24	7 BG	5.5/2	Grayish green
G	0.2923	0.3227	496	6	0.32	5 BG	6/2	Pale green
H	0.2986	0.3257	501	3	0.43	10 G	7/1	Light grayish green

\* Relative to component A.

ISCC-NBS system by use of the appropriate charts.<sup>23</sup> This permits the ready identification of these colors by the use of the Munsell Book of Color,<sup>21</sup> or the ISCC-NBS Dictionary of Color Names.<sup>23</sup>

The foregoing analysis was made to determine the spread of colors possible for the chosen standard subject. Unfortunately, the conditions of the analysis are difficult to obtain in the laboratory. Therefore, methods which approximate those conditions were chosen. Mono-energetic x-rays were approximated with narrow bands of x-rays obtained by selective absorption. Spectrally-pure light was approximated by narrow bands of light using a tungsten lamp and Wratten filters. In place of a white screen, color-sensitive photographic paper was used.

EXPERIMENTAL PROCEDURE—SEPARATION

Methods of obtaining narrow energy bands of x-rays involve the use of a diffraction grating, x-ray fluorescence, radioactive isotopes, or selective absorption.

The method of selective absorption was used in this study as the separation method. The freedom of varying the spectral distribution by changing the operating potential in conjunction with radiographic filters was not used in this study, since facilities were not available to measure the spectral distribution. It was necessary to start with

the known spectral distribution curve taken from the literature.

Selective absorption takes advantage of the fact that most materials are relatively transparent immediately adjacent to an x-ray absorption edge on the low energy side. Thus, by appropriate selection of type and amount of material, different narrow energy bands of x-rays can be obtained.

In this study, six elements were investigated as selective absorbers or radiographic filters. These were cadmium, tin, antimony,

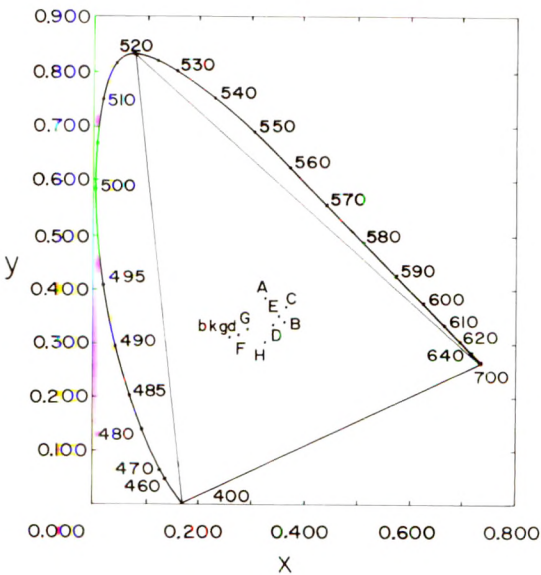


Fig. 7. Chromaticity plot of subject components.



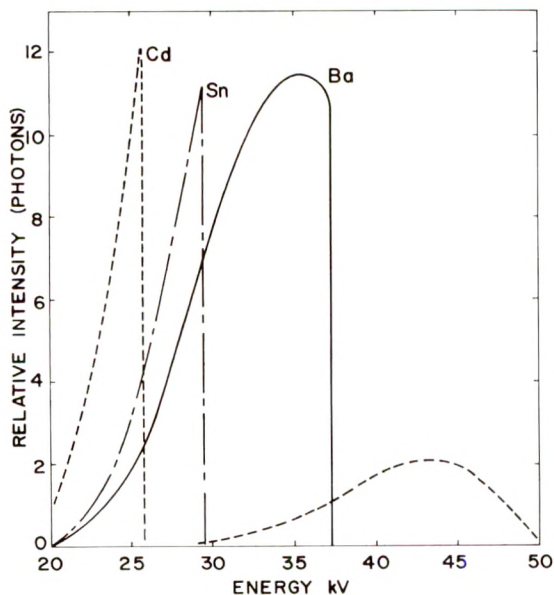


FIG. 8. Spectral distribution curves for Ba, Cd, and Sn filters.

barium, cerium, and lead. The spectral transmission curves were computed for each of these radiographic filters and these curves were multiplied by the original spectral distribution curve to obtain new spectral distribution curves. The new spectral distribution curves are shown in Figures 8 and 9, and the exposures necessary to obtain these curves are tabulated in Table v. Since the subject was inanimate, exposures could be made as long as necessary.

Radiographs were made of the subject with the above radiographic filters placed at the tube port. The new spectral distribution curves were multiplied by the spectral transmission curve of the subject and the spectral response curve of the film and new spectral stimulus curves were obtained for each subject component and radiographic filter. The areas under these new spectral stimulus curves compared favorably with measured densities.

Table vi shows the ratios of the subject component density to background density for the various radiographic filters.

From the data in the previous table, separation coordinates can be calculated analo-

gous to the chromaticity coordinates of the CIE system. The relative densities from a series of three separation radiographs can be treated as the tristimulus values are in the CIE system. Two combinations were analyzed in this manner. These were:

- $D = Cd$  filtered radiograph
- $E = Sb$  filtered radiograph
- $F = Ce$  filtered radiograph
- $G = Sb$  filtered radiograph
- $H = Ce$  filtered radiograph
- $I = Pb$  filtered radiograph

From the foregoing, the “separation coordinates” can be computed by the relationships:

$$d = \frac{D}{D + E + F} \quad e = \frac{E}{D + E + F}$$

$$f = \frac{F}{D + E + F}$$

Since  $d + e + f = 1$ , only two of these coordinates need be considered. For the other combination the coordinates were computed in the same manner. When two of these coordinates are plotted, “separation diagrams” are obtained analogous to a chromaticity diagram. These two diagrams are shown in Figure 10 and 11.

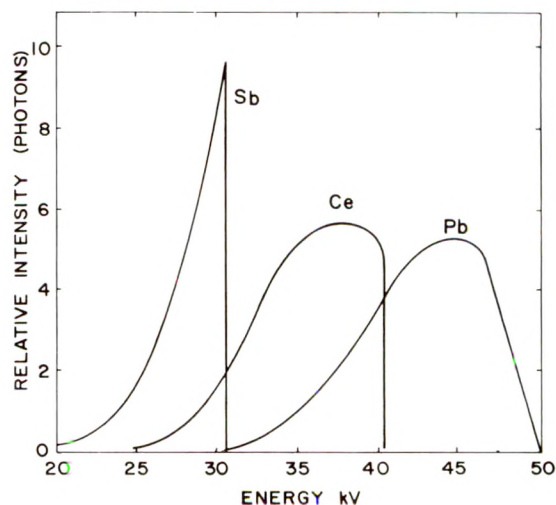


FIG. 9. Spectral distribution curves for Ce, Pb, and Sb filters.

TABLE V		
EXPOSURE FACTORS FOR RADIOGRAPHIC FILTERS		
Filter Type	Amount (mg./cm. <sup>2</sup> )	Exposure (mas.)
Cd	200	40
Sn	320	50
Sb	250	40
Ba	170	16
Ce	325	20
Pb	320	200

An indication of the color separation possible can be obtained from an examination of the "separation diagrams." The diagram, *g* vs. *h*, for the *Sb*, *Ce*, *Pb* combination, shows the subject components to fall in a more or less straight line. If this plot is superimposed on a chromaticity diagram, it can be seen that the color changes will be mainly changes in purity, or in hue over a relatively small range. The pattern of points in the diagram, *d* vs *e*, for the *Cd*, *Sb*, *Ce* combination, is more of an arc and if this were balanced around the neutral point in a chromaticity diagram, larger changes in hue could be obtained.

EXPERIMENTAL PROCEDURE—RECOMBINATION PROCESS

As stated before, color is not being reproduced in the radiograph, but is being introduced arbitrarily. This permits a large number of possible combinations. However,

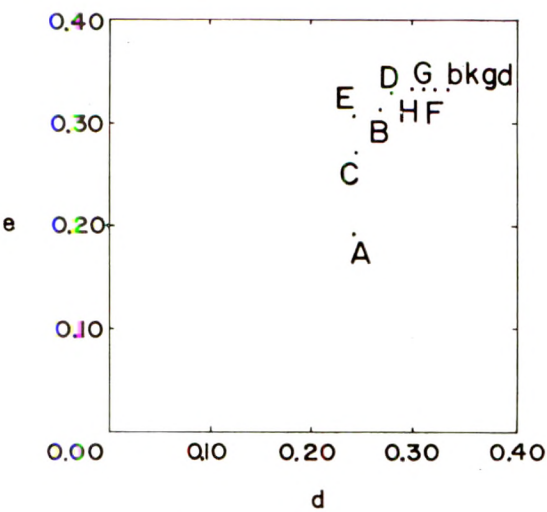


FIG. 10. Separation diagram—Cd, Sb and Ce.

one limitation is the fact that the eye is more sensitive to changes in hue than changes in purity or lightness. This means the distribution of colors should be balanced around the neutral point. The best method for recombination would be one that is very flexible. This could be some arrangement of three projectors, each able to illuminate its radiograph with a wide range of spectrum colors of various intensities. This type of an arrangement would permit the most information to be recovered. Such an arrangement would not give a permanent record in color, although the separation radiographs would be permanent.

TABLE VI						
RELATIVE TRANSMITTANCE OF PROCESSED RADIOGRAPHS						
Component	Filters					
	Cd	Sn	Sb	Ba	Ce	Pb
Background	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
A	0.0796	0.0450	0.0618	0.1071	0.1786	0.3394
B	0.3916	0.4054	0.4588	0.5306	0.6250	0.7459
C	0.1854	0.1712	0.2118	0.2832	0.3839	0.5528
D	0.4902	0.5315	0.5824	0.6301	0.6920	0.7683
E	0.2330	0.2469	0.3000	0.3622	0.4464	0.5569
F	0.7693	0.8108	0.8235	0.8380	0.8571	0.8720
G	0.6332	0.6577	0.6765	0.7002	0.7321	0.7581
H	0.4811	0.5135	0.5382	0.5638	0.5982	0.6402



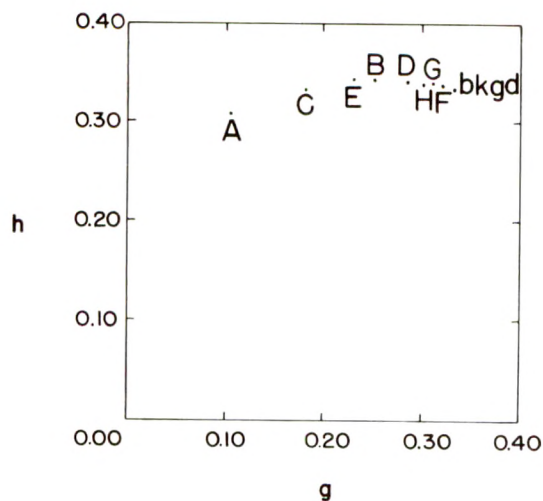


FIG. 11. Separation diagram—Sb, Ce and Pb.

The projection method for this study used a photographic enlarger in conjunction with Wratten colored filters.<sup>13</sup> The radiographs were projected in sequence onto color-sensitive photographic paper.

The photographic enlarger was a 4×5 inch Press Solar Autofocus Enlarger. This contains a G. E. 301 tungsten lamp which approximates the CIE standard Source A in its spectral distribution of light intensity.

The optical filters used were placed after the radiograph. These were Wratten Gelatin Filters No. 25A (red), No. 58 (green), No. 47B (blue).

The recording medium chosen for this portion of the study was Kodak Ektacolor Professional Paper. This choice was limited by the facilities available for processing color-sensitive paper. This is not necessarily the best color-sensitive paper for this purpose.

This type of paper is a reversal paper. The colors projected on it are recorded as complementary hues and bright areas become dark.

#### EXPERIMENTAL RESULTS

Two series of separation radiographs were analyzed. The first set consisted of separation radiographs taken with the Sb, Ce and Pb radiographic filters. When re-

combined, the components of the standard subject were recorded in various shades of brownish-reds and yellows. This fact confirmed the indication given by the combination's separation diagram. The second set consisted of separation radiographs taken with Cd, Sb and Ce radiographic filters. When this set was recombined, the components of the standard subject were recorded in various shades of blues, greens and yellows. This was also indicated by the combination's separation diagram.

#### DISCUSSION

The energy range covered in this study was limited by two factors. First, the film had not been evaluated much past 50 KV since the main motive in evaluating it was for the application in crystallography. Second, above 50 KV Compton scattering of photons becomes considerable and their attenuation cannot be predicted in a straightforward manner. If the energy range had included the normal range covered in radiography, the possibility for increased color would be greater.

The standard subject chosen had only eight components. If the range of densities and types of materials is extended, the possibility for increased color may be extended.

The change in color from one component to another is gradual since the change in the subjects' spectral transmission curve is gradual. The requirement for an abrupt change in color is that the component be highly opaque at one energy and highly transparent at another energy. This condition is approximated when an element has an absorption edge. The elements of organic molecules do not have an absorption edge in the energy range useful for radiography. However, many of the contrast media used in radiology, such as barium, iodine and silver, do have an absorption edge in the range of interest.

The method of approach followed in this study was chosen to facilitate analysis and is not necessarily the best method for pro-

ducing colored radiographs. The subject chosen was inanimate and no motion was involved. Exposures could be long and the film could be changed between exposures. This would not be the case for a live subject. However, it was necessary to separate the exposures to evaluate the effect of each independently. For clinical application it may be necessary to make the three separation negatives with one exposure. However, much more investigation is necessary before clinical application can be made.

This work is closely related to the original work in colored radiography done by Donovan.<sup>8,9,10</sup> However, there are several important differences. Donovan's work was largely empirical. The radiographic process was not analyzed. He made three exposures with the continuous unfiltered beam of the x-ray tube operated at 30, 32 and 34 KVP. Aitken and Dixon<sup>1</sup> show how the spectral distribution of x-ray photons varies with the applied voltage. From an examination of these data and the spectral transmission curve of the subject, it must be concluded that the separation was small on Donovan's three radiographs. Also in his recombination process, Donovan did not mention that the colors of the subject components should be distributed about the neutral point to bring out the maximum information. Others who applied Donovan's method (National Geographic Magazine, Sept., 1958) made an unbalanced image which is essentially monochromatic.

Donovan advocated the design of special equipment to make the three different exposures in rapid succession. Certainly the design of such equipment would not be difficult at the present level of technology but such an approach is premature. More study is needed before clinical application can be made.

Donovan did say that the projection method is the best method of recombination since much information is lost in color printing. It is our opinion that this loss of information can be more than overcome by substituting analytical methods for empirical methods in obtaining the colors.

#### CONCLUSION

Based on the results of this research in colored radiography, the following conclusions have been drawn:

1. The radiographic process can be completely defined by specifying (a) the spectral energy distribution curve of the x-ray source, (b) the spectral transmission curve of the subject, and (c) the spectral response curve of the recording medium. When these three curves are multiplied together, ordinate by ordinate, a spectral stimulus curve results. The measured densities on the processed radiographs are directly proportional to the areas under the spectral stimulus curves.

2. Each subject component has its own unique spectral transmission curve which is a function of its chemical composition, density and thickness. The magnitude and order of transparency change at different energies for the various subject components.

3. The x-ray beam can be resolved into narrow energy bands by selective absorption. When these narrow energy bands are used to take radiographs, the magnitude and order of the densities of the subject components are changed.

4. The possibility for colored radiography is an inherent property of the standard subject chosen for this study.

5. Colored radiographs do contain more information than conventional radiographs.

6. This study provides a basic analytical approach for the further study of colored radiography.

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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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*Forty-ninth Annual Meeting:* Royal York Hotel, Toronto, Ontario, Canada, May 29-31, 1967.



# EDITORIALS

## THE SIXTY-SEVENTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

FOR the first time in its history, the American Roentgen Ray Society convened in fabulous San Francisco, proverbially "everybody's favorite city." The occasion was the Sixty-seventh Annual Meeting, which was held at the new ultramodern San Francisco-Hilton Hotel from Tuesday, September 26 through Friday, September 30, 1966.

The meeting was called to order at 8:30 A.M. Tuesday morning by President John Paul Medelman of White Bear Lake, Minnesota. He introduced John B. de C. M. Saunders, M.B., F.R.C.S. (Edinburgh), M.D., F.A.C.D., Chancellor of the University of California Medical Center, San Francisco, California, who gave a warm Address of Welcome. Chancellor Saunders with eloquence cited fascinating episodes from the early history of San Francisco in which physicians played important roles, and narrated in some detail the tremendous advances in all phases of radiology which he witnessed at the University of California during the past 40 years. In paying his respect to the splendid work of radiologists, he extended cordial greetings and wished the meeting great success.

Chancellor Saunders' Address was a delightful prologue to the excellent Scientific Program which was to follow immediately after the installation of the new President.

Dr. Medelman then with the assistance of Dr. Herbert M. Stauffer, Chairman of the Executive Council, installed President-Elect Clyde A. Stevenson of Spokane, Washington, as the new President of the Society and presented him with the traditional Gold Medal of the President. He was also presented with the symbolic Pfahler gavel, fashioned from a mastodon's tooth recovered from Alaskan glacial ice, and the

working Erskine gavel made from memorabilia of Röntgen, Coolidge and Snook.

As a final part of the ceremony, incoming President Stevenson presented outgoing President Medelman with the Past-President's Book, inscribed with his name. This beautifully executed volume contains color photographs of the Pfahler and Erskine gavels, and a list of the past presidents of the Society.

After the conclusion of the induction into office, President Stevenson gave his Inaugural Address on "Problems of Radiology of Today and the Future." In this splendid Address, President Stevenson discussed the numerous problems existing in Radiology of today, some of which seriously threaten its future and need careful solution if the specialty is to survive. "Our strengths are few and our weaknesses many," said President Stevenson. The most important strength is "the full support of the American Medical Association and many specialty organizations, other than our own, in attempting to preserve our status as physicians practicing medicine in the specialty of Radiology." The prime weaknesses are the present shortage of radiologists and radiologic technologists in the face of increased demands for our services; an insufficient cooperative effort recognizing the opportunities and needs of both the academic and non-academic divisions of our specialty; persisting differences in the philosophy of postgraduate training; and many others. Furthermore, "the knowledge explosion in Radiology," has resulted in fragmentation into subspecialty branches, with more radiologists limiting their activity to a relatively smaller segment of the main specialty. President Stevenson also pleaded the cause of the "Generalists" in Radiology, who—in the

words of Millis—"to be competent, have to be brighter, better educated, more energetic and more experienced than the specialist." President Stevenson's most timely and thought provoking Address is published in full, as the leading article, in this issue of the JOURNAL.

The Scientific Program commenced at 9:10 A.M. Tuesday morning with President Stevenson in the Chair. Twelve papers were given at this morning's session, comprising such varied topics as: Para-endocrine Syndromes Associated with Carcinoma of the Lung; Percutaneous Transthoracic Needle Biopsy; The Thoracic Radiographic Manifestations of the Malignant Lymphomas; Asbestosis and Malignancy; The Chest Roentgenogram Following Supraglottic Sub-Total Laryngectomy; Pneumothorax and Pneumomediastinum in the Newborn Infant; Vascular Changes in Pulmonary Emphysema; The Lateral Film in Pulmonary Edema; Pneumocystis Carinii Pneumonia (Plasma Cell Pneumonia); Radiologic Manifestations of Confirmed Primary Tuberculosis Before and during Antimicrobial Treatment; Review of Logical Analysis Procedures for Roentgen Diagnosis; and Radioisotope Scanning of Extracranial Tumors.

As is customary, the Instruction Courses were scheduled for the afternoon. These courses, which for a long time have been an essential part of the Annual Meeting of the American Roentgen Ray Society, were again under the expert directorship of Dr. Harold O. Peterson, with Dr. Donn G. Mosser as Associate Director. An outstanding Faculty of Radiologists responded to the invitations of Drs. Peterson and Mosser and the material presented on the various aspects of radiology dealing with diagnosis, treatment and interrelated subjects attracted capacity audiences throughout the meeting.

Tuesday evening was reserved for the Caldwell Lecture, the most important scientific event of the Annual Meeting. This year the Society was in the uniquely fortunate situation of having one of the giants of contemporary medicine delivering this Lecture. He was introduced by F. J. L.

Blasingame, M.D., Executive Vice-President of the American Medical Association, Chicago, Illinois.

Dr. Blasingame in his "Introduction of Caldwell Lecturer of 1966" stated, "The speaker chosen to give the Caldwell Lecture tonight has been an effective man. His effectiveness has resulted from a generous combination of intelligence, energy, compassion, and honesty along with other characteristics which have brought him renown and respect. This effectiveness has been demonstrated in a number of ways: he has been an effective teacher; he has indelibly written his name across the gastrointestinal literature and has become nationally and internationally acclaimed for his knowledge and contributions concerning the medical aspects of colon disease; he has developed and used new knowledge which he has readily passed on to others through his treatment of patients, his contacts with his fellow physicians and others working in the health field, and through his extensive writings."

The illustrious Lecturer was J. Arnold Barga, M.D., Head of Section on Gastroenterology, Scott and White Clinic; and Director of Medical Education, Scott and White Memorial Hospital, Temple, Texas. At the end of Dr. Blasingame's introduction, Dr. Barga was given an unprecedented standing ovation by the distinguished audience of the evening. The title of his Lecture was "Chronic Ulcerative Colitis: Diagnostic and Therapeutic Problems. A Life Long Study."

At the conclusion of the Lecture, President Stevenson presented to Dr. Barga the Caldwell Gold Medal, the highest award of the Society for meritorious scientific achievement.

The "Introduction of Caldwell Lecturer of 1966" by Dr. Blasingame and the Lecture by Dr. Barga will be published in full in the January, 1967 issue of the JOURNAL.

On Wednesday morning with Dr. John F. Holt of Ann Arbor, Michigan, First Vice-President, in the Chair, the Scientific Program was continued. The following subjects were discussed: Cine-Cholangiomanometry; A Radiologic Approach to the



Renal Mass; Roentgenological Evaluation of Bladder Neck Obstruction; Contrast-Globulin Precipitation, A Possible Cause of Induced Renal Failure in Patients with Multiple Myeloma Undergoing Intravenous Pyelography; Fibromuscular Dysplasia of the Renal Arteries; Radiologic Observations on the Fate of Large Pulmonary Emboli; Pulmonary Embolic Disease—Pulmonary Angiography; Correlative Assessment of the Macroalbumin Lung Scan with the Clinical and Radiographic Chest Findings; Radiodiagnostic Techniques in the Diagnosis of Pulmonary Thromboembolism; Radioisotopic Techniques for Evaluation of Pulmonary Disease; Radioactive Scanning of Superior Mediastinal Masses; The Clinical Applications of the Anger Gamma Camera to Disease of the Thyroid; and Clinical Use of the Spark Imaging Camera in Nuclear Medicine.

On Wednesday afternoon at 1:30 P.M., the American College of Radiology presented a timely and extremely important Panel Discussion titled "A Report from the American College of Radiology," with Dr. Stevenson presiding. The members of the Panel were: Dr. J. E. Miller, Dallas, Texas, Moderator; Dr. Jackson E. Livesay, Flint, Mich.; Dr. Stanley M. Wyman, Cambridge, Mass.; and Dr. Antolin Raventos, Philadelphia, Pa., Panelists.

The Thursday morning Scientific Session was presided by Dr. Herbert M. Stauffer, Chairman of the Executive Council. There were 13 papers read at this session. They were: Biological Basis of Preoperative Radiation Therapy; Skin Sensitivity in Hyperbaric Radiation Therapy; The Paradox of Breast Cancer; Palliative Irradiation for Bladder Carcinoma; Carcinoma of the Palatine Tonsil; Strabismus Produced by Intracranial Extension of Cancer of the Nasopharynx can be Cured with Radiation Therapy; Complete Calibration of an X-Ray Machine; Vascular Jet Contraction in Angiography; Ten Years Experience in Celiac Arteriography in the Diagnosis of Pancreatic, Portal and Hepatic Pathology; Experience with Postmortem Cerebral An-

giography Performed as Routine Procedure of the Autopsy; Positive Contrast Demonstration of the Normal Auditory Canal; Pseudo-Suprasellar Tumors; and The Radiology of the Meningo-Hypophyseal Trunk.

Thursday evening, starting at 6:30 P.M., was the gala Social Affair of the Meeting. Preceded by a Cocktail Party in the Continental Parlors 7, 8 and 9, the Annual Banquet was held at 7:30 P.M. in the Continental Parlor 6. The highlight of this evening was the presentation of the Scientific Awards (described in the following pages of the JOURNAL). The banquet was followed by most enjoyable singing with orchestral entertainment and gay dancing.

The Friday morning Scientific Session, presided by Dr. Justin J. Stein of Los Angeles, California, Second Vice-President, included 11 papers, comprising the following topics: The Use of Barium Sulfate Suspension for the Purpose of Making Early Radiologic Diagnoses in Patients Suspected of Having Obstruction of the Small Intestine; Superficial Spreading of Carcinoma of the Stomach; The Retained Gastric Antrum; A Cartridge-Loaded Palm-Sized Syringe for Gas Contrast Radiography; Radiographic and Anatomic Features of Double Outlet Right Ventricle; The Predictive Value of Volumetric Study of the Left Ventricle; Biplane Selective Coronary Arteriography Via Percutaneous Transfemoral Approach; Controlled Exit Catheterization; The Newborn Skull; Amniography for Placental Localization; Intravenous Placentography; Sexual Precocity, Precocious Puberty and Virilism, Radiologic Findings; and A Roentgen Evaluation of Patients with the Turner's Phenotype.

The last series of Instruction Courses were given early Friday afternoon and were again completely sold out.

The Scientific Exhibits, assembled by Dr. Joseph O. Reed and his Committee; and the Technical Exhibits, authoritatively arranged by Mr. Clifford L. Sherratt, Honorary Member, and Dr. James C. Cook, Manager of the Annual Meeting of the Society, were numerous, of exceptional

quality and of highest artistic display. They contributed immeasurably to the great success of this year's Annual Meeting.

Yet, in a city of "auriferous splendor," reminiscent of the old gold dust days, the most significant factor contributing to the over-all success of a convention may perhaps be the genial hospitality of the local inhabitants derived from a rich heritage. Dr. Thomas M. Fullenlove, ubiquitous host as Chairman of the Committee on Local Arrangements, his charming wife, Sally, enthusiastic Chairman of the Ladies' Committee, and the very efficient members of their Committees, had arranged Social Activities of a most unique character.

On Monday, preceding the beginning of the meeting, the Annual Golf Tournament for Members and Guests was held at the famous Olympic Club of Lakeside, San Francisco, in beautiful surroundings, overlooking the serene Pacific Ocean. Competition was for the Willis F. Manges Trophy and the Exhibitors' Trophy. The local Golf Committee, under the Chairmanship of Dr. Walter W. Herbert, had made excellent arrangements which were greatly appreciated by the participants. The Tournament was followed by cocktails and the Annual Golf Dinner at the same Club.

On Monday afternoon the Ladies enjoyed a comprehensive tour of the city, including the Japanese Tea Garden in Golden Gate Park. This tour was concluded with the traditional Golf Widows' dinner, which was held at a restaurant in historical Ghirardelli Square, purchased by Ghirardelli, a young chocolate maker from Italy, in 1893, and redeveloped recently into a magnificent center of shops and restaurants. On this beautiful evening San Francisco was all rubies and diamonds, strewn across velvety hills, and the return to the hotel in roller coaster cable cars—officially declared an historic monument of San Francisco—was enchanting in its magical fascination.

Tuesday morning was reserved for a Conducted Behind-the-Scene Walking Tour of Union Square Shops, comprising San Francisco's most distinctive merchan-

dise centers, several of which are local landmarks.

On Wednesday, a bus trip was arranged into the world-famous Redwood Country. Crossing north on the Golden Gate Bridge to Marin County, the trip continued to Muir Woods, where a long stop allowed leisurely strolls through this 424 acre national monument, among the fabled California Redwoods in all stages of development. The Ladies then returned to Sausalito for lunch and in the afternoon visited the shops on the Sausalito waterfront.

Thursday morning was spent by visiting Chinatown, the largest Chinese settlement outside of Asia, and its many priceless treasures. The Ladies' tour ended at noon.

The Society is greatly indebted to Mrs. Thomas M. Fullenlove and her Committee for arranging this unusual Program and extends its sincerest thanks.

To the San Francisco Convention Bureau, and to the Management and Staff of the San Francisco-Hilton Hotel, the Society expresses its appreciation for the efficient registration, the excellent service and the many courtesies gracefully rendered throughout the meeting.

To Dr. Thomas M. Fullenlove, Chairman of the Committee on Local Arrangements, the Society owes special thanks for the suave way in which he, aided by the able members of his Committee, carried out every detail of the local arrangements.

The Sixty-seventh Annual Meeting held at San Francisco was generally acclaimed as the grandest in the history of the American Roentgen Ray Society. It had by far the largest attendance, its accommodations were superb, the Scientific Programs were conducted in a most congenial atmosphere, the Scientific and Technical Exhibits were unrivalled and the Social Activities were unparalleled. President Stevenson may take justifiable pride, for this was a convention that nobody who attended can forget. His radiant personality dominated with "auriferous" brightness the entire convention.

TRAIAN LEUCUTIA, M.D.





DOUGLAS A. QUICK

1891-1966

A LIFE of service to the cancer patient came to an end in Utica, New York, on January 30, 1966 when Dr. Douglas A. Quick passed to his reward.

Dr. Quick was born on November 10, 1891, on a farm one and one-half miles south of Harrow, Ontario, the son of Mr. and Mrs. Frederick Quick and of one of Essex

County's pioneer families. His primary and secondary education was obtained in Harrow schools. He graduated from the University of Toronto in 1914 with the degree of Bachelor of Medicine. After a locum tenens during the summer of 1914 he elected to move to New York for post-graduate training and to ultimately engage

in the practice of Surgery and Therapeutic Radiology. His initial appointment was at the City Hospital in New York in 1914. In 1916, he became an assistant to Dr. Henry Harrington Janeway, one of the leading radium therapists of his time. When Dr. Janeway died in 1921, Dr. Quick became an attending surgeon at Memorial Hospital, a post he held until 1932.

Working closely with the late Dr. Gioacchino Failla and with Dr. Edith Quimby, he developed techniques for the use of the  $\beta$  component of radium emanation (radon), first of all in the form of glass pearls for the irradiation of superficial basal cell carcinomata. Operating behind a shield which proved inadequate to filter out all of the gamma component of this form of radiant energy, and in darkened rooms, the treatment was given visually in contact fashion in "minutes" or "seconds" of exposure. Unfortunately, the hazards to the therapist were evidenced in later years when damage to the skin and bone of fingers resulted in the loss of some of these digits and seriously curtailed a busy surgical practice. Later, when Failla developed gold capillaries and radon seeds, the gamma component of radon came into its own particular field of usefulness as a source of interstitial irradiation, under the guidance of Quick and others.

Having worked with the 4 gram radium unit at Memorial Hospital, Dr. Quick had become convinced of the superiority of gamma irradiation over conventional orthovoltage techniques, particularly in the treatment of tumors of the head and neck. Therefore, when an opportunity was presented to Dr. Quick about the year 1947 to assist in the development of a 50 gram radium teletherapy unit, he became fascinated with the prospect and devoted much of his energy and resources to it. The fundamental design of the unit was worked out by Dr. Failla and took the form of a multiple source housing, each containing 2 grams of radium. The beam collimation was so arranged that each of the beams converged at a focal point, the theoretic tumor level,

and were dissipated in turn according to geometric principles. The unit was housed in the Janeway Clinic of the Roosevelt Hospital. The treatment of patients began in 1951 and proceeded day and night around the clock.\* Perhaps before the results of such treatment could be established, Kilocurie Cobalt 60 Teletherapy Units were developed in Canada and put in operation almost synchronously in Saskatoon, Saskatchewan and London, Ontario, with source strengths ranging to 1,300 curies and emitting pure gamma radiation at a fraction of the cost of radium. There can be no doubt, however, that Dr. Quick's project was contemporary and perhaps in advance of his time in light of the developments of succeeding years. The principles involved have been amplified by world wide experience with teletherapy and super-voltage systems.

Dr. Quick was active in many professional societies. He joined the American Radium Society as a member in 1919, three years after it was founded, became its President in 1926 at Dallas, Texas, and The Janeway Lecturer in 1937 on the subject, "Carcinoma of the Larynx," in New York City. He was a Trustee of the American Board of Radiology from 1939 to 1960 and its President in 1952. He joined the Radiological Society of North America in 1920, was its Carman Lecturer and Gold Medalist in 1947. He later became an Emeritus Member of the Radiological Society of North America and was also elected a Life Member of the American Roentgen Ray Society.

During the years, the writer had come to know Dr. Quick by virtue of association with Dr. N. A. McCormick of Windsor, who had worked with Dr. Quick in his private office in New York City. Some of the great respect that we hold for him in Essex County was derived naturally from this association. On occasion, we shared a social evening at one of the society meet-

\* QUICK, D., and RICHMOND, J. D. Preliminary experiences with a 50 gram converging beam radium unit. *AM. J. ROENTGENOL., RADIUM THERAPY & NUCLEAR MED.*, 1955, 74, 635-649.



## THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Sixty-seventh Annual Meeting of the American Roentgen Ray Society, held at the San Francisco-Hilton Hotel, San Francisco, California, September 27-30, 1966 were assembled by Dr. Joseph O. Reed, enthusiastic Chairman and his untiring Committee on Scientific Exhibits. They were the largest and most successful in the Society's history, depicting visually subjects of unusual variety, several of which complemented the scientific essays on the same subject presented at the Scientific Sessions.

The Exhibits were set up in the South-West and North Corridors of the Convention Floor, in the immediate vicinity of the room of the Scientific Assembly and of the Main Hall, housing the Technical Exhibits. This was a fortunate arrangement, permitting members and their guests to visit them frequently and to study them at leisure. The exhibits were of great documentative value and were displayed in a most attractive fashion.

The secret "Blue Ribbon" Committee on Awards—to use President Stevenson's accolade—after repeated deliberations and a most thoughtful consideration awarded one Silver Medal, one Bronze Medal and nine Certificates of Merit.

Following are brief descriptions of the exhibits:

### SILVER MEDAL

*Clinical Interpretation of the Lung Scan.* Philip M. Johnson, M.D., Kent Ellis, M.D., Bruce R. Parker, M.D., and Stuart W. Cosgriff, M.D., Columbia University College of Physicians and Surgeons, New York, New York.

This beautifully displayed exhibit presented the rationale, safety, clinical applications and diagnostic pitfalls of the pulmonary perfusion scanning (lung scan), based on more than 400 patient examinations.

The following technique of scanning was

usually employed by the exhibitors:

1. Block the thyroid with Lugol's solution, 10 drops t.i.d. for 4 days beginning 24 hours before scanning. For emergency scanning, give 100 mg. NaI intravenously followed by Lugol's solution for 3 days.

2. With the patient supine administer 300  $\mu$ c of a recently re-suspended solution of  $I^{131}$ MAA or 1-2 mc of  $Tc^{99m}$ MAA. The injection is made slowly while the patient breathes deeply in and out.

3. Find "hot spot" and begin scanning immediately. With high speed equipment scanning in one view requires 10-12 minutes and scanning in two views is performed whenever possible.

4. Obtain companion roentgenogram with the patient supine, at the end of shallow expiration at maximum target film distance.

5. The scanning may be repeated in 24 hours if necessary.

The  $I^{131}$ MAA was prepared according to a modification of the Taplin method. This modification was described in detail.

Selected clinical examples illustrated the unique value of the lung scan in detection and management of pulmonary thromboembolic disease and its role in the evaluation of patients with emphysema, neoplasia, congenital heart disease, pericardial effusion, subphrenic abscess and other diseases.

Conditions in which the clinical utility of the lung scan is impaired, such as congestive heart failure, mitral stenosis, etc., were also illustrated.

### BRONZE MEDAL

*The Topography of Carcinoma of the Paranasal Sinuses: Diagnostic and Therapeutic Implications.* Max L. M. Boone, M.D., Thomas S. Harle, M.D., Howard W. Higholt, M.D., and Gilbert H. Fletcher, M.D., The University of Texas, M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston, Texas.

Because of the complex bone anatomy of the paranasal sinuses, evaluation of the

extent of carcinomatous involvement is almost entirely dependent on detailed roentgenographic studies. Three-dimensional visualization of the anatomy of the disease, though difficult, is a necessary prologue to intelligent therapy.

Typical cases were presented to demonstrate the probable locations of primary growth and routes of spread for a full spectrum of involvement, from the earliest to the most advanced. The roentgenograms were supplemented with line drawings and three-dimensional models to illustrate the significant findings.

The details of appropriate treatment plans, including isodose distributions, were given along with the results and complications of radiotherapy.

#### CERTIFICATES OF MERIT

1. *Pediatric Case of the Day*. D. H. Altman, M.D., B. E. Bear, M.D., and R. E. Litt, M.D., Variety Children's Hospital and University of Miami, School of Medicine, Miami, Florida.

A different *Pediatric Case* was presented each day. On the basis of history and the pertinent roentgenograms exhibited only a single diagnosis was accepted.

The answers were given the following day with a discussion and demonstration in color of the pathology.

This exhibit, like the *Case of the Day* of Dr. Fullenlove, created an unusually great interest.

2. *Population Exposure to X-rays: U. S. Public Health Service X-ray Exposure Study*. Donald R. Chadwick, M.D., U. S. Public Health Service, Division of Radiological Health, Washington, D. C.

This exhibit was based on a Public Health Service study designed to provide information on the x-ray experience of the U. S. population.

The principal findings included estimates of the frequency and rate of persons examined and treated by x-rays, the number of various types of examinations and procedures, the volume of films related to radio-

graphic, fluoroscopic, and dental examinations, and indices of exposure associated with the several types of x-ray procedures.

3. *Asbestosis and Malignancy*. Nicholas G. Demy, M.D., and Howard Adler, M.D., The Somerset Hospital, Somerville, New Jersey.

The relationship between asbestosis and various neoplasms has been well documented in recent years.

The increased incidence of bronchogenic carcinoma, pleural mesothelioma, and peritoneal mesothelioma with concomitant asbestosis was illustrated. Proximity to a large asbestos manufacturing plant offered the unique opportunity to trace the sequential development of these varied tumors as observed clinically, roentgenographically, and pathologically. The basic roentgenographic changes were emphasized in cases of transition seen in asbestosis with superimposed malignancy. Gross and microscopic correlation of these tumors included isolation of asbestos bodies in neoplastic tissue.

The industrial and possible urban hazard of asbestos was discussed.

The authors also presented a scientific essay on the same subject.

4. *Case of the Day*. Tom M. Fullenlove, M.D., Saint Francis Memorial Hospital, San Francisco, California.

Cases which represent interesting and intriguing problems were exhibited daily. The answers were posted on the following day.

This year's cases of the day because of their unequalled excellence continuously attracted a very large number of radiologists.

5. *Paleo-Pathology—Pre-Columbian United States. Radiologic Interpretation of Diseases in Prehistoric Man*. Ronald R. Hagge, M.D., F.A.C.S., and Richard Wilkinson, B.A., M.A., Harper Hospital, Detroit, Michigan and University of Michigan, Anthropology Department, Ann Arbor, Michigan.

This extremely interesting exhibit presented numerous roentgenographic studies



of bone exemplifying the great value of this modality in determining diseased tissues, or the end result of disease processes, in the Pre-Columbian man, in the United States.

The following is the classification of the disease processes considered: *Trauma*: primary—accidental, fractures being the most common entity; secondary—induced, as for instance, head molding and trephination. *Infection*: non-specific—osteomyelitis, sinusitis, periostitis; specific—tuberculosis, syphilis, leprosy (no finally positive evidence has been found that these diseases existed Pre-Columbian, but more support is apparent). *Arthritis*: degenerative (osteoarthritis), frequently being confused with the end results of trauma; rheumatoid. *Tumor*: malignant (as studies increase, more evidence is being found)—primary, metastatic; benign. *Congenital Anomaly*: there was much anomalous change in the past as is in the present, perhaps, on the basis of close intermarriage of the hunting and gathering groups. *Deficiency Disease*: there is minimal evidence of this, perhaps, on the basis of an "eating off the earth" program. *Endocrine Disease*. *Miscellaneous*: calculi, fetal parts, unusual burial practices, mutilation, preservation of tissues other than bone, mummification (probably accidental and only in S.W.U.S.), Cu preservation (noted in 2,000 year old burials). *Dental Pathology*: the entire gamut of pathology of dental structures exists.

6. *Pulmonary and Pleural Lesions in Rheumatoid Arthritis*. William Martel, M.D., Murray R. Abell, M.D., William M. Mikkelsen, M.D., and Walter M. Whitehouse, M.D., University of Michigan Medical Center, Ann Arbor, Michigan.

The spectrum of pulmonary lesions in rheumatoid arthritis, based on observations in 35 patients was presented in this exhibit.

Discrete rheumatoid nodules, occasionally observed to cavitate, were seen in 12 cases and in 7 of these, their histologic nature was verified. Two patients developed spontaneous pneumothorax. These nodules may develop prior to arthritis and without an occupational exposure to pneu-

moconiosis. Three patients showed a transition from discrete nodules to diffuse interstitial pneumonitis, pulmonary fibrosis and persistent pleural effusion. Biopsy or autopsy material was available in 9 such cases. Bronchogenic carcinoma developed in 2 patients with pulmonary fibrosis.

7. *The American Institute of Radiology (AIR)*. Robert T. Morrison, American College of Radiology Foundation, Chicago, Illinois.

This exhibit gave the 1966 Interim Report of the AIR collections, and the inventory of them which now comprises 2,400 items and is categorized as follows. *Museum*: Accessories, 60; equipment, 40; tubes, 100; miscellaneous, 50. *Library*: Chairs, roentgen, 2; books, 400; catalogues, 60; journals, bound, 150 vol.; journals, unbound, 200; directories, 20; teaching manuals, 90; pamphlets, 150; programs, 165; reports, 250; reprints, 75; scrapbooks, 15. *Archives*: Charts, 10; cine films, 16; society histories, 20; photographs, 250; radiographs, 75; tape recordings, 4; documents, 350 folders (40,000 pages).

The exhibitor again stressed the need for continued support and interest of the radiological community.

8. *Decrease in Lymph Node Size Following Lymphangiography*. Richard J. Steckel, M.D., Radiation Branch, National Cancer Institute, Bethesda, Maryland.

Opacified lymph nodes appear to decrease in size roentgenographically over the months following lymphangiography.

Although treated pathologic lymph nodes often regress markedly on postlymphangiography roentgenograms, moderate decreases in lymph node size occur even in patients who have received no treatment. This fact is not always appreciated, and responses to therapy may be inferred from moderate changes in lymph node size when these changes are actually an artefact of lymphangiography.

The mechanism is probably that lymph nodes enlarge when ethiodol is injected and return to their pre-lymphangiographic size as the contrast medium is slowly

eluted. Clinical material and experimental studies on dogs illustrated these points.

9. *Vascular Changes in Pulmonary Emphysema*. A. Franklin Turner, M.D., George Jacobson, M.D., and Edward A. Behnke, M.D., University of Southern California School of Medicine and Los Angeles County General Hospital, Los Angeles, California.

This exhibit described the vascular changes associated with chronic obstructive pulmonary disease (emphysema) demonstrated by various roentgenologic techniques including plain roentgenography, tomography, selective pulmonary angiography, single film and cone wedge arteriography.

Six cases were presented in detail illustrating the range of changes which may be encountered. These are: differential blood flow; dilatation of the main pulmonary artery and its major branches with increased attenuation of the larger peripheral arteries; reduction in the number of smaller peripheral arteries, arterioles and veins; obliteration of the capillary bed; and arteriovenous shunts. The extent of involvement may vary greatly from one segment of lung to another with one area being normal and the other markedly abnormal.

The plain roentgenograms of the chest in patients with emphysema may be normal. Hyperinflation and air-trapping are non-specific findings and may be entirely absent. Reduction in the peripheral pulmonary vessels, the most reliable evidence of emphysema, may not be detectable on the chest roentgenogram even though advanced vascular changes are present.

Selective pulmonary and peripheral pulmonary wedge angiography provide the only roentgenologic means of assessing, with a reasonable degree of accuracy, the vascular damage in patients with emphysema. Knowledge of the extent of permanent vascular damage is of great importance in determining the prognosis which can be offered the patient and the improvement which may be expected from therapeutic measures.

#### OTHER EXCELLENT EXHIBITS WERE:

*Complete Diagnostic Evaluation of the Infant Cardiac, Under Fifteen Pounds*. Adele R. Altman, M.D., Robert E. Kahn, M.D., Arnold J. Slovis, M.D., and Frank J. Borrelli, M.D., Departments of Radiology and Pediatrics, New York Medical College—Flower and Fifth Avenue Hospitals, New York, New York.

This exhibit presented an opportunity for comparing formalin-fixed hearts with the patient's clinical findings, chest roentgenograms, hemodynamic data, and angiocardio-grams.

Representative examples of widely variable anatomic complexes were drawn from a series of 125 patients.

The indications for complete cardiac workup and the low rate of complications were stressed. The exhibitors' present technique of using sodium diatrizoate 50 per cent for angiocardiology in infants was described.

*Pancreatic Scanning*. A. Rodriguez-Anunez, M.D., E. J. Filson, M.D., S. O. Hoerr, M.D., R. E. Hermann, M.D., C. H. Brown, M.D., B. H. Sullivan, Jr., M.D., F. J. Owens, M.D., and E. I. Winkelman, M.D., Cleveland Clinic Foundation, Cleveland, Ohio.

The exhibit consisted of 4 panels presenting: (1) a brief clear explanation of the principles of pancreatic scanning; (2) three normal pancreatic scans illustrating the methods of interpretation and means of recognizing important landmarks on the scans; (3) six abnormal pancreatic scans, all surgically verified, where recognition of tumor is obvious; and (4) a statistical report of the exhibitors' experience in over 160 patients who underwent pancreatic scanning.

*Diagnosis and Operative Treatment of Aorto-Iliac Occlusive Disease*. Howard C. Baron, M.D., Department of Surgery, Jewish Memorial Hospital, New York, New York.

The exhibit illustrated steps in diagnosis and operative treatment of atherosclerotic vascular disease of the terminal aorta and



iliac arteries. The operative technique of endarterectomy of the aortoiliac segment with aortograms illustrating the arterial pathology was shown.

A brief review of the indications for operation with summary of results obtained was included.

*The Newborn Pelvis: Normal and Abnormal Patterns.* N. Ray Baumgartner, M.D., University of Cincinnati Medical Center, Cincinnati, Ohio.

The roentgenographic appearance of the newborn pelvis does not resemble closely its anatomic counterpart. The margins of the pelvis are composed of cartilage, possessing a radiodensity similar to that of the surrounding soft tissues. The true outline of the pelvis is therefore obscure, since only the ossified portions are visualized on the roentgenogram.

Photographs and roentgenograms of the dissected newborn pelvis were used to illustrate the cartilaginous component and were compared with antemortem roentgenograms.

Serial roentgenograms from birth to 10 years of age demonstrated the growth and ossification of the pelvis.

The diagnostic features of the pelvis found in mongolism, achondroplasia, and congenital dislocation of the hip were described and illustrated.

*Conventional Tomography of the Lungs in Bronchogenic Carcinoma: An Approach to Diagnosis and Prognosis and Correlation with Bronchoscopy.* Irwin Bluth, M.D., and Bruce Steger, M.D., The Brookdale Hospital Center, Brooklyn, New York.

Tomography of the lungs with cancer offers 4 advantages over conventional roentgenography. (1) The lesion in the lung can be better studied for circumscription, calcification and cavitation. (2) Involvement of the air-outlined bronchi is more apparent. (3) Distortions of the vascular pattern associated with peripheral cancers are more readily shown. (4) Finally, invasion of the mediastinum is more easily appreciated.

Conventional roentgenograms with cor-

responding tomograms were presented to demonstrate these 4 points.

*A New Approach to Colon Preparation for X-Ray Examination.* Garland R. Brown, M.D., University of Michigan Medical Center, Ann Arbor, Michigan.

A new approach to cleansing the colon for roentgenologic examination of the colon or other abdominal structures was described utilizing a combination of special diet, magnesium citrate and bisacodyl.

Analysis of a large controlled study showed excellent results and high patient acceptance.

Procedure details, statistical data and typical roentgenograms were presented.

*Venography in the Subfertile Male with Varicocele.* Jordan S. Brown, M.D., Lawrence Dubin, M.D., Melvin H. Becker, M.D., and Robert S. Hotchkiss, M.D., Departments of Urology and Radiology, New York University School of Medicine, New York, New York.

This exhibit displayed an original technique for performing internal spermatic venography, which has been done on 32 subfertile men with varicoceles.

The venous anatomy of the testicle was shown; the possible etiology of varicocele was illustrated; and the relationship of varicocele to subfertility was discussed.

*The Newborn Skull.* Charles N. Chasler, B.S., M.D., University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania.

Approximately 2,000 roentgenographic examinations of the newborn skull were reviewed at the Magee-Women's Hospital and Children's Hospital in Pittsburgh, Pennsylvania from 1962 to 1965.

Unusual congenital anomalies of the bones of the skull were carefully studied and the most illustrative cases were selected for demonstration. The subject of trauma to the newborn skull was also intensively reviewed. Each case was closely scrutinized from the standpoint of a complete history, both from the obstetrical and pediatric viewpoints.

Definite conclusions could be obtained from these studies in reference to particu-

lar radiolucent lines that could be interpreted as fractures. It was also decided that at times a definite roentgen diagnosis could not be made. The importance of a very thorough history and physical examination was definitely ascertained.

The exhibitor also had a scientific essay on the same subject.

*The Arterial Collateral Network in the Pelvis: An Arteriographic Study.*

Arnold Chiat, M.D., Arnold Moltz, M.D., and James H. Nelson, M.D., Kings County Hospital Center, Downstate Medical Center, S.U.N.Y., Brooklyn, New York.

Spontaneous pelvis arterial anastomoses occur following occlusion of pelvic arteries by disease processes.

The exhibitors demonstrated by means of percutaneous transfemoral arteriography: (1) that these anastomoses occur following ligation of *normal* arteries; (2) that in the normal, anastomoses occur as an *immediate* sequel to ligation; (3) that in the light of this it is virtually impossible to devascularize a vascular lesion in this region; and (4) that occlusion of major vessels during the course of catheterization may pass unnoticed and perfusion remain adequate.

The exhibit contained 7 illustrative cases and a description of the pertinent arterial anatomy.

*The Renogram—Diagnostic and Prognostic Features Based on Proved Cases.* Max D. Clark, M.D., Lucille A. DuSault, A.B., Eugene J. Kochkodan, M.D., and William R. Eyler, M.D., Henry Ford Hospital, Detroit, Michigan.

Analysis of renograms from patients with proved diagnoses of nephrosclerosis, nephritis, and renal artery disease, indicates features essential in diagnosis and prognosis for surgical relief.

The importance of an exacting technique was stressed.

*Mammography in Clinical Practice.* James T. DeLuca, M.D., and John H. Wentworth, M.D., The Community Hospital at Glen Cove, Glen Cove, New York.

The exhibit delineated the x-ray findings

and diagnosis in association with the clinical indications responsible for the examination.

From 5,000 problematic patients examined by mammography, a summary of the first 175 cancers encountered disclosed occult cancers of the breast at the rate of 3 per 1,000 problematic patients. The 37 per cent axillary involvement was below the national average reported by other groups and is most encouraging. Many other benefits derived through the application of mammography in the diagnostic armamentarium of the radiologist were summarized.

*Magnification Lymphography.* Theodore Ditchek, M.D., and Gerard T. Scanlon, M.D., Yale University School of Medicine, New Haven, Connecticut.

In an attempt to increase the accuracy of diagnostic lymphography, magnification of the lymph nodes and channels opacified by ethiodol was performed.

A specially designed and constructed roentgen-ray tube was utilized to obtain very sharp images at 3 times linear (9 times area) magnification.

The normal and involved lymph nodes in individuals with a wide spectrum of neoplastic and inflammatory diseases were studied. The magnification studies frequently showed the lymph nodes to be normal or abnormal when the diagnosis was unclear on conventional lymphangiograms. The features of the normal lymph node architecture were better delineated.

Subclinical pulmonary oil microemboli were demonstrated in a large number of patients.

*Angiographic and Hemodynamic Studies in Pulmonary Embolism.* Josef Edelstein, M.D., Stephen N. Wiener, M.D., and Bernard L. Charms, M.D., Mt. Sinai Hospital, Cleveland, Ohio.

The value of pulmonary angiography and hemodynamic data following right heart catheterization, as important and complementary diagnostic studies, was shown by the results obtained in 68 patients referred with a tentative diagnosis of pulmonary embolism.



The angiographic involvement of larger and smaller arteries was exhibited.

The response of the pulmonary artery pressure to unilateral pulmonary artery balloon occlusion was studied in 19 of these patients. An attempt was made to correlate the response to the extent and nature of embolic involvement.

*Mammography and Breast Cancer.* Robert L. Egan, M.D., R. Waldo Powell, M.D., and James V. Rogers, M.D., Emory University School of Medicine, Atlanta, Georgia.

The problem of breast cancer was presented, emphasizing: occurrence, spread and salvage rate; indication for mammography; and importance of physician team approach.

Practical exercises in clinical examination, mammography and study of gross and microscopic pathology in breast diseases were included.

*70 MM. Filming.* (A) *Evaluation of Some Physical Factors Affecting Quality.* (B) *Examples of Routine and Special Usage.* Richard A. Elmer, M.D., Crawford W. Long Memorial Hospital of Emory University, Atlanta, Georgia.

Some of the physical factors affecting quality were evaluated. These comprised: (1) several types of films, all with identical processing; (2) results of several methods of processing, including several standard automatic processors; (3) resolution of a lined phantom and comparable (of the same patient) with an 0.3 mm. focal spot, 1.2 mm. focal spot, and conventional spot roentgenograms; (4) geometric magnification and lens magnification utilizing over-framing with 300 mm. lens as compared with 70 mm. films exposed with a standard lens (210 mm.).

Examples of clinical application included: (1) usage in routine fluoroscopic examinations; and (2) usage in unusual situations to illustrate some of the potential of this modality.

*Factors in the Selection of a Contrast Medium for Angiography.* Harry W. Fixher, M.D., Dale E. Fuller, M.D., and Steven

H. Cornell, M.D., University of Iowa Hospitals, Iowa City, Iowa.

Iodine content, viscosity and toxicity are all highly important in the rational selection of an angiographic contrast medium.

The little appreciated and poorly understood characteristics of the commonly used contrast media were presented in graphic form. No agent currently in use is perfect, but the angiographer who knows the opacification which is desired can make a rational decision on the choice of a contrast medium without sacrificing safety.

*A Cooperative Evaluation of Mammography in Seven Teaching Hospitals.* Adele K. Friedman, M.D., Samuel I. Askovitz, M.D., Simon M. Berger, M.D., Gerald D. Dodd, M.D., Mary S. Fisher, M.D., Marc S. Lapayowker, M.D., Jeffery P. Moore, M.D., Donald E. Parlee, M.D., Goerge N. Stein, M.D., and Eugene P. Pendergrass, M.D., University of Pennsylvania, Philadelphia, Pennsylvania.

This exhibit summarized the results of a cooperative study of 2,000 consecutive mammograms.

Before mammography the clinical diagnosis of each breast was recorded; 776 breasts were biopsied.

The roentgenographic diagnostic accuracy (true positive rate) in "clinically obvious carcinoma" was 81 per cent but in patients with "dominant masses not obviously carcinoma" the true positive rate was 50 per cent. Histologically the yield of malignancy in patients with no dominant mass was 12/1,000 of which 6/1,000 were correctly diagnosed roentgenographically.

Mammograms illustrating typical mammographic problems were displayed. An evaluation of the usefulness of mammography in the clinical management of breast problems was presented.

*Ultrasonic Aortography.* Barry B. Goldberg, M.D., Bernard J. Ostrum, M.D., and Harold J. Isard, M.D., Albert Einstein Medical Center, Philadelphia, Pennsylvania.

Ultrasonic angiography is a safe rapid

technique whereby the abdominal aorta can be outlined and its lumen diameter, wall thickness and over-all size accurately measured. In addition, it can detect sites of occlusion. The procedure is painless and inexpensive and, if necessary, can be performed at the bedside.

No other single technique offers these advantages. It can be used to determine the precise location of an aneurysm and to differentiate it from a solid or cystic mass. Elderly individuals suspected of harboring aneurysms can be readily screened and those patients with aortic dilatation can be closely observed by serial studies.

The exhibit amply demonstrated these advantages.

*Pseudo Suprasellar Tumors.* Wm. Hanafee, M.D., L. Bilodeau, M.D., J. Alberti, M.D., and G. Wilson, M.D., University of California Medical Center, Los Angeles, California.

Spurious shadows such as fluid collections or the rectus gyrus of the frontal lobe may closely simulate tumors in the suprasellar region.

Tomography in the translateral and erect positions will eliminate the confusing shadows and allow a much more accurate delineation of the anatomy of the suprasellar region.

The exhibitors presented a scientific essay on the same subject.

*The Crescent Pattern of Increased Radioactivity in Brain Scanning.* William J. Heiser, M.D., James L. Quinn, III, M.D., and William V. Mollihan, M.D., Chicago Wesley Memorial Hospital, Chicago, Illinois.

The crescent pattern of increased radioisotope activity seen in the anterior or posterior projection of brain scanning was thought to be specific for subdural hematoma. As experience in brain scanning has increased, the exhibitors found this pattern to be present in a variety of pathophysiologic states.

Total integration of the patient's signs and symptoms along with the results of other ancillary laboratory tests permits

the interpreter to approach the specific diagnosis of this scan pattern more intelligently. Not infrequently one must still rely upon cerebral arteriography or craniotomy for more definitive diagnosis.

Illustrations of this scan pattern in patients with subdural hematoma, granulomatous pachymeningitis, scalp trauma, craniotomy defect, cerebral vascular accident, and metastatic carcinoma were included.

*New Concepts of Cerebral Angiography with Special Emphasis on New Techniques of Catheterization in the Geriatric Patient.* Sadek K. Hilal, M.D., Ph.D., Ernest H. Wood, M.D., and Bernard L. Grossman, M.D., Columbia-Presbyterian Medical Center, New York, New York.

In view of the known risk involved in the angiographic study of the patient suffering from cerebrovascular disease the exhibitors have developed a technique of aortic catheterization which is believed to be best suited for the examination of the geriatric patient suffering from cerebrovascular disease. A critical appraisal of the roentgenographic quality of the films obtained was presented with special attention to the visualization of the intracranial collateral circulation.

Isopaque 440 was the contrast medium injected. Its advantages and the optimum conditions of its use were studied in animals and in the clinical material presented. The results indicate that the concept of the "contact time" of the contrast material with the vessel wall is particularly important when this new contrast agent is administered. This finding resulted in the modification of the automatic injector used.

Further improvements on the aortic catheterization technique include a new design of an arterial puncture needle, a new guide wire, and a new modification of the rapid film changers and tube stands which makes possible the biplane visualization of all the cerebrovascular system from the aorta upwards with one injection made in the aortic arch, thus reducing the quantity of opaque medium administered and the



length of the procedure, both of which are factors affecting the rate of complication.

*Total Arteriography—A Fundamental Concept.* W. J. Howland, M.D., W. M. Burns, M.D., and J. L. Curry, M.D., Ohio Valley General Hospital, Wheeling, West Virginia.

This exhibit stressed the importance of opacifying arteries from their aortic origin to their termination in most angiographic studies. Emphasis was placed on preliminary aortography and retrograde arteriography in order to provide a "road map" prior to selective or subselective arteriography.

Clinical applications were shown which point up the need for this simple, safe and efficient basic approach.

Illustrative examples included cerebral, mesenteric, renal, extremity and other angiographic studies.

*Selective Coronary Arteriography.* Melvin P. Judkins, M.D., University of Oregon Medical School, Portland, Oregon.

This exhibit described the technique of selective coronary arteriography performed by the transcutaneous femoral approach, using a re-shaped Cordis catheter.

The method was successfully employed in 85 cases. One of its main advantages is that the patient can be turned with the catheter *in situ* for filming in various projections.

Typical left and right arteriograms were presented.

*Selective Angiography of the Adrenal Gland.* Paul C. Kahn, M.D., Carl E. Cassidy, M.D., and Lester V. Nickrosz, M.D., Tufts-New England Medical Center and Veterans Administration Hospital, Boston, Massachusetts.

Complete selective angiographic visualization of the adrenal glands is now possible, by a method combining selective inferior phrenic artery injection, selective middle adrenal artery injection and angiography of the renal and celiac arteries following intra-arterial epinephrine.

The exhibit summarized the technique

employed and the findings in approximately 40 studies.

Examples of angiograms were shown of normal variations, adrenal hyperplasia, adrenal adenoma, and adrenal metastatic tumor, with correlation of the radiologic and pathologic appearance of the gland in a number of cases.

*Tomography of the Ear: A Study of Malformations.* Leon G. Kaseff, M.D., Graduate Hospital, University of Pennsylvania, and Presbyterian-University of Pennsylvania Medical Center, Philadelphia, Pennsylvania.

Tomography is indispensable in the diagnosis and planning of corrective surgery in congenital ear malformations. Clinical evaluation and hearing tests often give no indication as to the development of the deformed ear. Highly precisioned pluridirectional tomography can best demonstrate the pathologic anatomy of the temporal bone. This preoperative information is necessary in patients with atresia or stenosis of the external auditory canal, middle ear deformities and first branchial arch malformations.

Examples of these malformations were demonstrated with complete clinical, audiometric, tomographic and surgical correlation.

*Lung Cancer Cavity and Differential Diagnosis.* Seuk Ky Kim, M.D., Department of Radiology, Duke University Hospital and Veterans Administration Hospital, Durham, North Carolina.

In the past it was thought that most lung cavities are caused by infection. However, in recent years it was found that an appreciable number of lung cavities are due to cancer. Similar to the situation of solitary pulmonary nodule, cavitation of the lung has become a difficult diagnostic problem in differentiating between cancer and infection. How does the cancer cavity differ from an infectious cavity? What type of malignancy cavitates?

The exhibit illustrated and summarized the findings in this differential diagnosis on the basis of many cases of cancer cavity

which were compared with infectious cavities.

*The Diagnosis and Evaluation of Therapy of Cerebrovascular Disease by Retrograde Brachial Angiography.* Robert A. Kuhn, M.D., New Jersey College of Medicine, Jersey City, New Jersey.

This exhibit was designed to illustrate the valuable role of retrograde brachial angiography in the diagnosis and localization of cerebral vascular disorders and the application of this method to the evaluation of the therapeutic management of such disorders.

A description of the procedure and the equipment used was offered along with a method of determining the velocity of cerebral blood flow.

Roentgenograms of patients with cerebrovascular disturbances demonstrating the use of this method in the objective evaluation of a vasodilating drug on cerebral perfusion were presented.

*Renal Arteriography.* Leon Love, M.D., Rogelio Moncada, M.D., and Carlos J. Reynes, M.D., Cook County Hospital, Chicago, Illinois.

In a large county hospital there is an opportunity of seeing some unusual renal vascular lesions.

The exhibitors presented illustrative cases of renal arterio-venous fistula, trauma, tumor and pseudo-tumor, and congenital absence of kidney.

*The Hepatic Angle in the Roentgen Evaluation of Peritoneal Fluid.* Milton Margulies, M.D., and Leonard Stoane, M.D., State University of New York, Downstate and Kings County Hospital, Brooklyn, New York.

Much of the difficulty encountered in determining the presence or absence of free intraperitoneal fluid can be eliminated by evaluating the anatomy of the right flank using the inferolateral border of the liver, the *hepatic angle*, as a reference point. A clearly defined hepatic angle denotes the absence of intraperitoneal fluid in this region; an obliterated angle (with preservation of the immediate extraperitoneal fat)

indicates free fluid in the peritoneal cavity. Colonic contents do not significantly alter the usefulness of this sign.

The exhibitors presented documentary evidence to prove the value of this sign.

*The Clinical Application of the Anger Gamma Camera to Diseases of the Thyroid.* John D. McAllister, M.D., Gilbert H. Alexander, M.D., Harold E. Goldberg, M.D., and Joseph A. Marasco, Jr., M.D., St. Francis General Hospital, Pittsburgh, Pennsylvania.

The Anger gamma camera is a relatively recent development in Nuclear Medicine. Its chief value lies in the speed with which excellent scans can be obtained. While extreme versatility is possible with the camera, the thyroid by reasons of its superficial location and anatomy does not require any special views.

The exhibitors described their work on phantoms indicating that resolution is essentially the same as that seen in conventional scanning. Normal thyroid glands were illustrated showing wide variation in size and configuration. Abnormal thyroid glands were also included demonstrating hyperthyroidism, adenomas both "hot" and "cold," thyroiditis and carcinoma.

The exhibitors gave a scientific essay on the same subject.

*The Quantitation of Differential Renal Clearance without Ureteral Catheterization.* I. Meschan, M.D., William Boyce, M.D., H. E. Schmid, M.D., C. Douglas Maynard, M.D., F. C. Watts, B.S., and R. L. Witcowski, M.S., Bowman-Gray School of Medicine, Winston-Salem, North Carolina.

This exhibit presented a summary of: (1) renal clearances of renografin- $I^{131}$ ; (2) technique and methodology of performing the procedure; (3) representative normals and abnormals; and (4) the exhibitors' experiences to date with performance of these differential clearances in various pathogenic states in graphic form.

*Chronic Pulmonary Diseases in Infants and Children.* Victor G. Mikity, M.D., and Peter Kane, M.D., Children's Division, Los Angeles County General Hospital and



University of Southern California, Los Angeles, California.

Chronic pulmonary disease in children can present difficult problems in diagnosis.

Congenital diseases, such as lobar emphysema, idiopathic pulmonary hemosiderosis, delayed pulmonary maturity of the premature, juvenile Hamman Rich syndrome and bronchopulmonary sequestration were demonstrated.

Acquired diseases of both infectious and noninfectious origin were also illustrated.

The important pathologic changes and diagnostic features of each entity were emphasized.

*Variations of the Petro-Sagittal Angle and Its Importance in Diagnostic Roentgenology.* Harold J. Peggs, M.D., Kaiser-Permenente, Santa Clara, California.

One thousand skulls of the TODD collection were compared anthropometrically and roentgenographically and confirmed on 100 live patients. All data were computer analyzed, the figures being subjected to  $R^2$  and  $T^2$  accuracy tables.

In essence the exhibitor found that two-thirds of humans have petro-sagittal angles which differ plus or minus degrees.

The exactness of all studies approaching the petron axially (usually Mayer and Owen) are directly dependent upon these angles. Prior measurement anthropometrically or by basilar view of the skull can determine these angles exactly.

*Clivus Chordomas: Report of Four Cases.* H. F. Plaut, M.D., and Ethyl S. Blatt, M.D., Veterans Administration Center and Veterans Administration Hospital, Dayton and Cincinnati, Ohio.

Chordomas are rare tumors being observed most commonly in the sacrococcygeal region. They occur in the base of the skull in about 35 per cent of cases, are invasive and destructive but do not metastasize.

This exhibit emphasized the rate of growth, asymmetric development and calcification which seems to be more frequent than formerly reported.

*Radiologic Findings in Cerebral Gigan-*

*tism.* Andrew K. Poznanski, M.D., and John N. Stephenson, M.D., Henry Ford Hospital, Detroit, Michigan.

Cerebral gigantism is a syndrome consisting of symptoms of excessively rapid advancement in stature and weight with increased maturity. In addition, special cranio-facial morphology and mental retardation are present in varying degree.

The exhibit consisted of a complete clinical, endocrine, cytogenetic and roentgenologic evaluation of 9 children seen at Henry Ford Hospital who have had characteristics of this syndrome.

The roentgenologic findings include a disproportionate maturity of the phalangeal epiphyseal ossification centers as compared to the maturation of the carpal or tarsal centers, thickening of soft tissues in some of the patients, and occasionally evidence of ventricular dilatation.

*Arteriography in Adrenal Tumors.* Plinio Rossi, M.D., St. Vincent's Hospital, New York, New York.

Several representative cases illustrating the value of arteriography were demonstrated. These included adrenal tumors—primary or metastatic—as shown by selective arteriography.

*Roentgenologic Evaluation of Bladder Neck Obstruction.* Charles E. Shopfner, M.D., Children's Mercy Hospital, Kansas City, Missouri.

The roentgen features of bladder neck obstruction were correlated with vesicoureteral reflux, bladder trabeculation, and residual urine in 527 children.

Circumferential contractures and anterior and posterior defects of the bladder neck are manifestations of degree of urethral distention, position of the bladder base plates, and urethral configuration. The bladder neck diameter was not the narrowest point of the bladder outflow tract in any patient.

This exhibit demonstrated that there is no consistent correlation of the roentgen features with vesicoureteral reflux, bladder trabeculation and residual urine. Contractures, defects, and diameter measurements

of the bladder neck are not valid roentgen criteria of bladder neck obstruction.

*Osseous Manifestations of Leukemia in Children.* Carole R. Simmons, M.D., Thomas S. Harle, M.D., and Edward B. Singleton, M.D., Baylor University College of Medicine and Texas Children's Hospital, Houston, Texas.

Leukemic involvement of bone often presents diagnostic features and at times may produce osseous changes simulating various other skeletal disorders.

This exhibit included examples of the various types of osseous changes which demonstrate the common and unusual manifestations of leukemia in children.

*Femoro-Tibial Bypass.* Renate L. Soulen, M.D., R. Robert Tyson, M.D., and Dominic A. DeLaurentis, M.D., Temple University Medical Center, Philadelphia, Pennsylvania.

The increasing incidence of peripheral vascular disease and the technical advances in vascular surgery have made arterioplastic procedures widespread. Small vessel surgery, however, has been largely limited to laboratory animals.

The exhibitors' experience with reconstructive surgery utilizing autogenous vein grafts to small vessels below the knee has demonstrated: (1) the absolute necessity of accurate preoperative arteriograms to assess operability and determine surgical approach; (2) the technical feasibility of such surgery in humans; and (3) the ability of any one of the 3 major vessels of the lower leg to sustain the extremity.

Roentgenologic and surgical methods and selected cases were shown.

The exhibitors also had a scientific essay on the same subject.

*Arteriography in Orthopedics.* T. W. Staple, M.D., R. G. Evens, M.D., and A. H. Stein, Jr., M.D., Mallinckrodt Institute of Radiology and Department of Orthopedics, Washington University School of Medicine, St. Louis, Missouri.

The exhibit was a survey of the techniques and indications for arteriography in orthopedic problems.

A central panel illustrated these points. Sixteen representative roentgenograms demonstrated its use in trauma, inflammatory and neoplastic disease and postoperative situations.

*Roentgenologic and Clinical Evaluation of Vesicoureteral Reflux and Pyelonephritis in Children.* Alfred L. Weber, M.D., Walther T. Weylman, M.D., Thomas E. Shea, M.D., and Guy W. Leadbetter, M.D., Massachusetts General Hospital, Boston, Massachusetts.

This exhibit dealt with vesicoureteral reflux in the pediatric age group.

The etiology of reflux was discussed briefly.

Representative cases were illustrated to evaluate the diagnosis of reflux on the intravenous urogram and cinecystogram. Special emphasis was placed on reflux and its relationship to pyelonephritis. Cases followed for several years were presented to show the deleterious effects of reflux. The increased incidence of reflux in patients with double ureters and collecting systems was illustrated. The behavior of reflux during drug therapy, following vesical neck surgery and urethrotomy, was evaluated.

A concise section on treatment of vesicoureteral reflux completed the exhibit.

*Angiography in Musculo-Skeletal Trauma.* Mark H. Wholey, M.D., Sam Poller, M.D., Klaus M. Bron, M.D., John M. Hendrickson, M.D., and H. Paul Newman, Departments of Radiology, University of Pittsburgh School of Medicine, The Veterans Administration Hospital, and Allegheny Valley Hospital, Pittsburgh, Pennsylvania.

The application of angiography in thoracic, abdominal and peripheral arterial injuries associated with combined musculoskeletal trauma was illustrated. Information of this nature is essential to the proper surgical approach.

*Amniography for Placental Localization.* George T. Wohl, M.D., John P. Emich, M.D., Samuel Wiltchik, M.D., Myron Blumberg, M.D., and Richard H. Schwarz,



M.D., Philadelphia General Hospital, Philadelphia, Pennsylvania.

Placental localization is frequently of major importance in determining proper management of patients with third trimester bleeding. Amniography, a simple technical procedure in which a water soluble contrast material is injected into the amniotic sac and appropriate roentgenograms are taken, is a new technique of high accuracy which permits easy and positive interpretation.

The results of placental localization in 50 patients in whom this technique was applied were demonstrated.

The incidental findings of interest relating to amniography were also incorporated into the exhibit and the exhibitors pre-

sented a scientific essay on the same subject.

*Roentgen Diagnosis of Unilateral Exophthalmos.* Judah Zizmor, M.D., Carl Fasano, M.D., Byron Smith, M.D., and William Rabbett, M.D., Departments of Radiology, Ophthalmology and Otolaryngology, Manhattan Eye, Ear and Throat Hospital, New York, New York.

Roentgen signs of unilateral exophthalmos are evident on routine roentgenograms in about 50 per cent of the cases.

This exhibit demonstrated that pneumotomography, opaque orbitography, phlebography or arteriography may indicate the cause of exophthalmos when routine roentgenograms are unrevealing.



## THE TECHNICAL EXHIBITS

THE TECHNICAL EXHIBITS of the Sixty-seventh Annual Meeting of the American Roentgen Ray Society, held September 27-30, 1966 in San Francisco, California, were unsurpassed in every respect. The ultramodern facilities of the luxurious San Francisco Hilton Hotel, besides providing an inviting atmosphere of a resort, made possible the concentration of all activities of the Society (with the exception of a few Instruction Courses) on the Convention Floor. The main Exhibit Hall, comprising joined Continental Parlors 1, 2, 3, 4 and 5 was located side by side with the Ballroom, comprising joined Continental Parlors 6, 7, 8 and 9, where the Scientific Sessions were held. Overflow exhibits were placed in the East-North and South Corridors, in continuation with the South-West and North Corridors, where the Scientific Exhibits were set up.

This uniquely convenient arrangement, together with the fact that the Program Committee, under the able Chairmanship of President-Elect Clyde A. Stevenson, programmed all Scientific Sessions in the morning and Dr. Harold O. Peterson, Director of the Section on Instructions, had scheduled all Instruction Courses in the afternoon, afforded the members of the Society and their guests, attending this year's Annual Meeting in record number, unexcelled opportunities to visit these exhibits. They took advantage of this to the fullest extent and were richly rewarded, for they were able to view a dazzling array of items magnificently displayed. These included a great assortment of new x-ray, isotopic and ultrasound equipment, accessories, x-ray tubes, image amplifiers, gamma and photo cameras, projectors, films, screens, automatic processors, improved catheters and injectors, chemicals and radiopharmaceuticals, the latest publications in books and encyclopedias, and many other items of current interest.

As in past years, Mr. Clifford L. Sherratt, Honorary Member, and Dr. James C.

Cook, Manager of the Annual Meeting, spared no time or effort in making all the detailed preparations for the exposition of these Exhibits, thereby enhancing immeasurably the over-all success of this year's meeting. The Society wishes to thank them most sincerely.

The Society also wishes to express its profound appreciation to the following firms for their valuable contribution to the over-all success of the Annual Meeting:

Abbott Laboratories, North Chicago, Illinois; Agfa-Gevaert, Inc., Teterboro, New Jersey; Albert Acan X-Ray Solutions, Inc., Detroit, Michigan; Americana Corporation, Beverly Hills, California; Atomic Energy of Canada Limited, Ottawa, Ontario; Auto-typist, San Francisco, California; Automatic Seriograph, Division of Litton Industries, College Park, Maryland; Barber-Colman Company, Rockford, Illinois; Barnes-Hind Barium Products, Sunnyvale, California; Bar Ray Products, Inc., Brooklyn, New York; Cenco X-Ray Company, Chicago, Illinois; Cook, Incorporated, Bloomington, Indiana; Continental X-Ray Corporation, Chicago, Illinois; Cordis Corporation, Miami, Florida; Dunlee Corporation, Bellwood, Illinois; Eastman Kodak Company, Rochester, New York; E. I. Du Pont de Nemours & Company, Wilmington, Delaware; Encyclopaedia Britannica, Chicago, Illinois; Eureka X-Ray Tube Corporation, Chicago, Illinois; E-Z-EM Company, Inc., Port Washington, Long Island, New York; General Aniline & Film Corporation, New York, New York; General Electric Company, X-Ray Department, Milwaukee, Wisconsin; Gordon Consultants, Inc., New York, New York; Great Books of the Western World, Chicago, Illinois; Grune & Stratton, Inc., New York, New York; Halsey X-Ray Products, Inc., Brooklyn, New York; Hoeber Medical Division, Harper & Row, Publishers, New York, New York; Hoechst Pharmaceuticals, Inc., Cincinnati, Ohio; Hoffrel Instruments Incorporated, Nor-



walk, Connecticut; Hogan X-Ray Company, Philadelphia, Pennsylvania; Ilford Inc., New York, New York; Keleket, Division of Laboratory for Electronics, Inc., Waltham, Massachusetts; Lea and Febiger, Philadelphia, Pennsylvania; Liebel-Flarsheim Company, Cincinnati, Ohio; Machlett Laboratories, Incorporated, Springdale, Connecticut; Mallinckrodt Pharmaceuticals, St. Louis, Missouri; N & H Instruments, Inc., Carrollton, Texas; North American Philips Co., Inc., New York, New York; Nuclear-Chicago Corporation, Des Plaines, Illinois; Pako Corporation, Minneapolis, Minnesota; Physicians Technical Equipment Company, Inc., New York, New York; Picker X-Ray Corporation, Cleveland, Ohio; Polaroid Corporation, Cambridge, Massachusetts; Profex-ray, Inc., Division of Litton Industries, Maywood, Illinois; The Purdue Frederick Company, Yonkers, New York; W. B. Saunders Company, Philadelphia, Penn-

sylvania; Schick X-Ray Company, Inc., Chicago, Illinois; Frank Scholz X-Ray Corporation, Boston Massachusetts; Siemens Medical of America Inc., Union, New Jersey; Smith Kline Instruments Company, Philadelphia, Pennsylvania; E. R. Squibb & Sons, New York, New York; Sperry Products, Division of Automation Industries, Inc., Danbury, Connecticut; J. W. Stacey, Inc., Palo Alto, California; Standard X-Ray Company, Chicago, Illinois; Charles C Thomas, Publisher, Springfield, Illinois; U. S. Catheter & Instrument Corporation, Glens Falls, New York; U. S. Radium Corporation, Morristown, New Jersey; Walker Industries, San Francisco, California; Westinghouse Electric Corporation, Baltimore, Maryland; The Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Year Book Medical Publishers, Inc., Chicago, Illinois.



## NEWS ITEMS

### NEW OFFICERS OF THE AMERICAN ROENTGEN RAY SOCIETY

At the Sixty-seventh Annual Meeting of the American Roentgen Ray Society held at the San Francisco-Hilton Hotel, San Francisco, California, September 27-30, 1966, the following officers were elected: *President*: Dr. Clyde A. Stevenson, Spokane, Washington; *President-Elect*: Dr. C. Allen Good, Rochester, Minnesota; *1st Vice-President*: Dr. Arthur E. Childe, Winnipeg, Manitoba, Canada; *2nd Vice-President*: Dr. Edgar M. McPeak, Washington, D.C.; *Secretary*: Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Georgia; *Treasurer*: Dr. Stephen W. Brown, 1467 Harper Street, Augusta, Georgia 30902.

The new Chairman of the Executive Council is Dr. J. Scott Dunbar, Montreal Children's Hospital, 2300 Tupper Street, Montreal, P.Q., Canada.

The Sixty-eighth Annual Meeting of the Society will be held at the Washington-Hilton Hotel, Washington, D.C., September 26-29, 1967.

### SOCIETY FOR PEDIATRIC RADIOLOGY

The Ninth Annual Meeting of the Society for Pediatric Radiology was held September 26, 1966, in conjunction with the Sixty-seventh Annual Meeting of the American Roentgen Ray Society, at the San Francisco-Hilton Hotel, San Francisco, California.

The following officers were elected: *President*: Dr. Harvey White, Chicago, Illinois; *President-Elect*: Dr. Martin H. Wittenborg, Boston, Massachusetts; *Secretary-Treasurer*: Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Boulevard, Los Angeles, California 90027.

The Tenth Annual Meeting of the Society will be held at the Washington-Hilton Hotel, Washington, D. C., September 25, 1967.

### AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS

The membership of the American Club of Therapeutic Radiologists has overwhelmingly voted for a change in the name of their organization to the "American Society of Therapeutic Radiologists."

A new Constitution and By-Laws have been adopted.

The Board of Directors of the Society is composed as follows: Milford D. Schulz, *Chairman*; James J. Nickson, *President*; Henry S. Kaplan, *President-elect*; Simon Kramer and Melvin L. Griem, *Members-at-Large*; Howard B. Latourette, *Treasurer*; and Juan A. del Regato, *Secretary*.

The Members of the Society will convene twice annually upon the occasions of the Annual Meetings of the Radiological Society of North America and the American Radium Society.

The first Scientific Session of the American Society of Therapeutic Radiologists was held in conjunction with the Annual Meeting of the Radiological Society of North America in Chicago, on November 29, 1966.

### ARMED FORCES INSTITUTE OF PATHOLOGY (AFIP)

Lieutenant Commander Elias Paul George Theros, MC, USN, recently was appointed the new Chief of the Registry of Radiologic Pathology at AFIP. He succeeds Colonel William L. Thompson, MC, USA (Ret.), who had served as Chief of the Registry since its founding in 1947.

The eminence of this Registry today is due almost entirely to the work of Colonel Thompson. He is most noted for his selfless dedication to bettering the instruction of radiology. Students came from countries throughout the world to attend his classes. He firmly believed that personal contact was far better than any written word.

For his outstanding contributions to the



field of radiology, Colonel Thompson was awarded the Scroll of the Radiological Society of North America in December, 1965, and the Gold Medal of the American College of Radiology in February, 1966.

#### POSTGRADUATE SEMINAR "CURRENT STATUS OF GENITOURINARY ROENTGENOLOGY"

A Postgraduate Seminar on "Current Status of Genitourinary Roentgenology," sponsored by the University of Miami School of Medicine will be held at the Fontainebleau Hotel, Miami Beach, Florida, March 20-23, 1967.

The Seminar will emphasize the current trends in genitourinary roentgenology. A total of 38 papers, two panel discussions and two film reading sessions will be presented.

The following topics will be covered: (1) radiologic examination of the urinary tract; (2) radiologic pyelonephritis; (3) renal papillary necrosis; (4) obstructive and non-obstructive hydronephrosis; (5) nephrocalcinosis; (6) renovascular hypertension; (7) radiologic diagnosis of renal tumors and cysts; (8) tumors of the renal pelvis and ureter; (9) exotic lesions of the upper and lower urinary tract; (10) renal vein thrombosis; (11) radiologic and pathologic studies of the adrenal glands; (12) kidney trauma; (13) ureteral pathology (notching, stricture, retroperitoneal fibrosis, congenital anomalies); (14) low ureteric obstructions in infancy and childhood; (15) ureteral reflux; (16) cystourethrography; (17) obstructive urethropathies; (18) vesical neoplasms; (19) trigonal canal; (20) genitography; (21) urologic procedures and homotransplantation; and (22) current status of special procedures in obstetric and gynecologic roentgenology.

In addition to the local Faculty, the following distinguished Guest Speakers will participate in this Seminar: Dr. Reed Altemus, Pittsburgh, Pennsylvania; Dr. John L. Emmett, Rochester, Minnesota; Dr. John A. Evans, New York, New York; Dr. Benjamin Felson, Cincinnati, Ohio;

Dr. C. J. Hodson, F.R.C.P., F.F.R., London, England; Dr. Thomas F. Meany, Cleveland, Ohio; Dr. Meyer M. Melicow, New York, New York; Dr. Harry Z. Mellins, Brooklyn, New York; Dr. Alphonse J. Palubinskas, San Francisco, California; Professor Ulf Rudhe, Stockholm, Sweden; and Dr. Charles E. Shopfner, Kansas City, Missouri.

The Director of the Seminar is Dr. Raymond E. Parks, Chairman, Department of Radiology, University of Miami School of Medicine; the Chairman of the Scientific Program Committee is Dr. Manuel Viamonte, Jr., and the Chairman of the Registration Committee is Dr. Robert F. Feltman.

For further information please write to Department of Radiology, University of Miami School of Medicine, Jackson Memorial Hospital, Miami, Florida 33136.

#### POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY—THE URINARY TRACT

The Department of Radiology of the New York Hospital—Cornell Medical Center will give a Postgraduate Course in "Urinary System of Children" April 20-22, 1967, in the Cornell Medical College Auditorium, 1300 York Avenue (at 69th Street), New York City, New York.

Complementing to local Faculty, the distinguished Guest Faculty comprises Dr. Donald H. Altman, Miami, Florida; Dr. David H. Baker, New York, New York; Dr. Walter E. Berdon, New York, New York; Dr. John P. Dorst, Baltimore, Maryland; Dr. J. Scott Dunbar, Montreal, P.Q., Canada; Dr. Leonard M. Freeman, New York, New York; Dr. Bertram R. Girdany, Pittsburgh, Pennsylvania; Dr. Edith L. Potter, Chicago, Illinois; Dr. Charles V. Pryles, New York, New York; and Dr. James A. Wolff, New York, New York.

For application for enrollment please write to Herman Grossman, M.D., Department of Radiology, New York Hospital—Cornell Medical Center, 525 East 68th Street, New York, New York 10021.

## NEW YORK ROENTGEN SOCIETY

The Spring Conference of the New York Roentgen Society will be held during Thursday, April 27; Friday, April 28; and Saturday, April 29, 1967, at the Waldorf Astoria Hotel in New York.

For further information please contact Arnold L. Bachman, M.D., Secretary, New York Roentgen Society, Francis Delafield Hospital, 99 Fort Washington Avenue, New York, New York 10032.

VIIIth SYMPOSIUM  
NEURORADIOLOGICUM

The VIIIth Symposium Neuroradiologicum will be held in Unesco Building, Paris, September 25th to September 30th, 1967.

Further information may be obtained from Professor H. Fischgold, Hôpital de la Pitié, 83 Boulevard de l'Hôpital, Paris XIIIème, France.

THE R. J. REEVES RADIOLOGICAL  
SOCIETY

The R. J. Reeves Radiological Society held a meeting on Saturday, November 12th, honoring Dr. Reeves, Professor of Radiology at Duke University, on the occasion of his retirement as administrative head of the Department of Radiology.

The major event of the meeting was the evening address by Dr. Ross Golden, preceded by a cocktail party and dinner.

As part of the weekend, an announcement was made that the R. J. Reeves Society and the Department of Radiology are planning to honor Dr. Reeves with an oil portrait and that the new departmental library will be named for Dr. Reeves.

Dr. Reeves was one of the early radiologists in North Carolina. He has built a teaching program at Duke University, from which approximately one hundred residents have received their training.





## BOOK REVIEW AND BOOKS RECEIVED

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*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

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**RADIOLOGIC DIAGNOSIS IN INFANTS AND CHILDREN.** By Armand E. Brodeur, M.D., M.R.D., F.A.C.R., Chief Radiologist, Cardinal Glennon Memorial Hospital for Children, St. Louis, Mo.; Associate Professor of Radiology and Associate Dean, St. Louis University School of Medicine, St. Louis, Mo.; Medical Director, School of X-ray Technology, St. Louis University, St. Louis, Mo.; Chairman, Committee on Public Education, American College of Radiology; Consultant to the Division of Radiological Health of the United States Public Health Service and The Catholic Hospital Association of the United States and Canada. Cloth. Pp. 503, with 757 illustrations. Price, \$26.50. The C. V. Mosby Company, 3207 Washington Boulevard, St. Louis, Mo., 1965.

In 1945 Dr. John Caffey published his magnificent text on *Pediatric X-ray Diagnosis*, and this book was revised in 1950, 1956, and 1961.\* Since the field of pediatric radiology is expanding and advancing so rapidly, there is a real need for another general textbook in the subspecialty of Pediatric Radiology. However, such an undertaking is so monumental that one may question whether a comparable work will ever again appear; the task may be too overwhelming or the subject too large, for any one author or for a single book. Regrettable as it seems, postgraduate students in radiology may have to depend increasingly upon monographs on individual body systems or groups of diseases.

Doctor Brodeur's single volume of *Radiologic Diagnosis in Infants and Children* only partially meets the needs of those to whom he has addressed it—"medical students, interns, residents, pediatricians, general radiologists, pathologists, and physicians orientated to pediatrics . . ."

One of the most obvious and serious weaknesses of the book is its lack of adequate references to the literature. Over 200 references are listed at the back of the book, but without any attempt to refer to this bibliography through-

out the text. Thus, the reader is given no guidance if he wishes to read more widely on any subject discussed, or to check the authority for any of the statements made. For example (Page 53), the author states "The Mayo series, reporting calcification in 15 percent of all tumors of the brain, generally agrees with our experience." What Mayo series? If the reader already knows, he has presumably little need for this text. If he does not, but wishes to find a reference for the "Mayo series," he must be prepared to go through the complete bibliographic list, and even then there is no assurance that he will find the "Mayo series" so casually mentioned.

The section on the mandible is written by a dental surgeon, but the roentgenographic reproductions are often disappointing and the language sometimes rather vague; there is a reference, for example, on Page 85 to a "pathologic abscess condition."

As regards the problem of radiation hazard to the infant and child, Doctor Caffey, in the preface to the fourth edition of his textbook, stated that "the most pressing current problem in pediatric radiology is the reduction of hazards to the patient from exposure to ionizing radiation. Pediatric radiologists must become deeply interested in the problems of improving protection for the patient . . ." This aspect is not sufficiently stressed in Doctor Brodeur's book and indeed many of the reproduced roentgenograms demonstrate exposure of more of the patient than would seem desirable for the diagnostic information being sought.

J. SCOTT DUNBAR, M.D.

### BOOKS RECEIVED

**ORTHOPÄDISCHES DIAGNOSTIKUM: BILDER, ÜBERSICHTEN, TABELLEN.** By Dr. med. Hans U. Debrunner, Aarau/Schweiz. Paper. Pp. 128, with 98 illustrations. Price, kartoniert DM 25.-. Georg Thieme Verlag, Stuttgart. In U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y., 1966.

**VIRUSES INDUCING CANCER: IMPLICATIONS FOR THERAPY.** Edited by Walter J. Burdette, A.B., A.M., Ph.D., M.D., Professor of Surgery and

\* The Fifth Edition is in press.

- Associate Director, M. D. Anderson Hospital and Tumor Institute, The University of Texas, Houston, Texas. Cloth. Pp. 498, with some illustrations. University of Utah Press, Salt Lake City, Utah, 1966.
- PRIMARY HEPATOMA.** Edited by Walter J. Burdette, A.B., A.M., Ph.D., M.D., Professor and Head of the Department of Surgery and Director of the Laboratory of Clinical Biology, University of Utah College of Medicine; Surgeon-in-Chief, Salt Lake County Hospital; Chief Surgical Consultant, Veterans Administration Hospital, Salt Lake City, Utah. Cloth. Pp. 150, with some illustrations. University of Utah Press, Salt Lake City, Utah, 1965.
- LEGAL CONSIDERATIONS ON IONIZING RADIATION: RADIONUCLIDES AND RADIATION EMITTING DEVICES.** By Gerald L. Hutton, LL.B., Assistant to the Director, Division of Safety Standards, U. S. Atomic Energy Commission, Washington, D. C. Cloth. Pp. 93. Price, \$5.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1966.
- ALPHABETICAL INDEX OF ROENTGEN DIAGNOSES AND PROCEDURES: WITH CODE NUMBERS OF THE AMERICAN COLLEGE OF RADIOLOGY.** A Cross-index of Roentgen Diagnoses Alphabetized for a Variety of Filing Systems. By Gerhart S. Schwarz, M.D., Chief of Radiologic Services, New York Medical College Chronic Disease Center; Bird S. Coler Hospital; Professor of Radiology, Flower & Fifth Avenue Hospitals; Visiting Radiologist, Metropolitan Hospital, New York, N. Y.; and Henry J. Powsner, M.D., Major, U.S.A.F., M.D., Chief of Radiology Service, 7520th United States Air Force Hospital, South Ruislip, Middlesex, United Kingdom. Pocket Edition. Cloth. Pp. 204. Price, \$8.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1966.
- PERIPHERAL ARTERIAL DISEASE.** By Wiley F. Barker, M.D., Professor of Surgery, University of California, Los Angeles, School of Medicine. Volume IV in the Series, Major Problems in Clinical Surgery. J. Englebert Dunphy, M.D., Consulting Editor. Cloth. Pp. 229, with some illustrations. Price, \$8.50. W. B. Saunders Company, West Washington Square, Philadelphia, 1966.
- COMPUTERS IN MEDICINE BIBLIOGRAPHY.** Edited by Arch H. Turner, Jr., and Dean A. Schmidt. Published by the Department of Radiology and the Medical Center Library, School of Medicine, University of Missouri, Columbia, Mo. Paper. School of Medicine, University of Missouri, Columbia, Mo., 65201, August, 1966.
- TEXTBOOK OF RADIOTHERAPY.** By Gilbert H. Fletcher, M.D., Radiotherapist, Professor of Radiology, Head of Department of Radiotherapy, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas. Cloth. Pp. 580, with 367 illustrations. Price, \$20.00. Lea & Febiger, 600 S. Washington Square, Philadelphia, 1966.
- THE DEVELOPMENT OF ZONAL CENTRIFUGES AND ANCILLARY SYSTEMS FOR TISSUE FRACTIONATION AND ANALYSIS.** National Cancer Institute Monograph 20, June, 1966. Edited by Norman G. Anderson, Ph.D., Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn. Cloth. Pp. 526, with some illustrations. Price, \$4.75. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 1966.
- STRAHLENSCHÄDEN UND STRAHLENHÄMATOLOGIE.** Vorträge aus einem Fortbildungslehrgang 1964 der Deutschen Akademie für ärztliche Fortbildung. Cloth. Pp. 166, with some figures and tables. Price, Geb. MDN 25,—. Veb Verlag Volk und Gesundheit, Berlin, 1965.
- DEUTSCHER RÖNTGENKONGRESS 1965.** Bericht über die 46. Tagung der Deutschen Röntgengesellschaft vom 29. April bis 2. Mai, 1965, Nürnberg. Paper. Pp. 236, with 347 illustrations. Price, DM 73,—. Georg Thieme Verlag, Stuttgart 1, Germany. In U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y., 1966.





## SOCIETY PROCEEDINGS

### MEETINGS OF RADIOLOGICAL SOCIETIES\*

#### UNITED STATES OF AMERICA

##### AMERICAN ROENTGEN RAY SOCIETY

*Secretary*, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Washington-Hilton Hotel, Washington, D. C., Sept 26-29, 1967.

##### AMERICAN RADIUM SOCIETY

*Secretary*, Dr. Fernando G. Bloedorn, Division of Radiotherapy, University of Maryland Hosp., Baltimore, Md. 21201. Annual meeting: Royal York Hotel, Toronto, Ont., Canada, May 29-31, 1967.

##### RADIOLOGICAL SOCIETY OF NORTH AMERICA

*Secretary-Treasurer*, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 27-Dec. 2, 1966.

##### AMERICAN COLLEGE OF RADIOLOGY

*Executive Director*, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Century Plaza Hotel, Los Angeles, Calif., Jan. 31-Feb. 4, 1967.

##### SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION

*Secretary*, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit, Mich. 48201. Annual meeting: Atlantic City, June 18-22, 1967.

##### AMERICAN BOARD OF RADIOLOGY

*Secretary*, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Fall 1966 examination will be held at the Washington Hilton Hotel, Washington, D.C., December 5-9, inclusive. The deadline for filing applications was June 30, 1966.

The Spring 1967 examination will be held at the Pittsburgh Hilton Hotel, Pittsburgh, Pennsylvania, June 5-9, inclusive. The deadline for filing applications for this examination is December 31, 1966.

The Fall 1967 examination will be held at the Statler Hilton Hotel, Dallas, Texas, December 4-8, inclusive. The deadline for filing applications is June 30, 1967.

##### AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE

*Secretary*, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

##### AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS

*Secretary*, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo. 80907

##### AMERICAN SOCIETY FOR DIAGNOSTIC ULTRASOUND

*Secretary*, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

##### TWELFTH INTERNATIONAL CONGRESS OF RADIOLOGY

*President*, Dr. Kempo Tsukamoto, 9-1, 4-chome, Angewa, Chiba, Japan. Meeting: Hotel New Otane, Tokyo, Japan, Oct. 6-11, 1969.

##### NINTH INTER-AMERICAN CONGRESS OF RADIOLOGY

*Counselor for the United States*, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

*President*, Dr. Leandro Zubiaurre, Montevideo, Uruguay. Meeting: Hotel San Rafael, Punta-del Este, Uruguay, Nov. 29-Dec. 5, 1967.

##### INTER-AMERICAN COLLEGE OF RADIOLOGY

*President*, Dr. Oscar Soto, H. Urteaga 480, Lima, Perú.

##### ALABAMA RADIOLOGICAL SOCIETY

*Secretary*, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

##### AMERICAN NUCLEAR SOCIETY

*Treasurer*, Raymond Maxson, 86 E. Randolph St., Chicago, Ill. Annual meeting to be announced.

##### AMERICAN SOCIETY FOR DIAGNOSTIC ULTRASOUND

*Secretary*, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

##### ARIZONA RADIOLOGICAL SOCIETY

*Secretary-Treasurer*, Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

##### ARKANSAS CHAPTER OF AMERICAN COLLEGE OF RADIOLOGY

*Secretary-Treasurer*, Dr. William J. Rhinehart, St. Vincent Infirmary, Little Rock, Ark. 72205.

##### ARKANSAS RADIOLOGICAL SOCIETY

*Secretary*, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

##### ASSOCIATION OF UNIVERSITY RADIOLOGISTS

*Secretary-Treasurer*, Dr. Stanley Rogoff, Department of Radiology, University of Rochester Medical School, Rochester, New York 14620. Annual meeting: Temple University School of Medicine, Philadelphia, Pa., May 12-13, 1967.

##### ATLANTA RADIOLOGICAL SOCIETY

*Secretary*, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

##### BAVARIAN-AMERICAN RADIOLOGIC SOCIETY

*Secretary*, Colonel Kurt Harrell, Landstuhl Army Medical Center, Landstuhl, Germany. Meets quarterly.

##### BLOCKLEY RADIOLOGICAL SOCIETY

*Secretary-Treasurer*, Dr. R. John Gould, 441 Lombardy Rd., Drexel Hill, Pa. 19026.

##### BLUEGRASS RADIOLOGICAL SOCIETY

*Secretary-Treasurer*, Dr. Arthur Lieber, University of Kentucky, University Hospital, Lexington, Kentucky. Meets quarterly.

##### BROOKLYN RADIOLOGICAL SOCIETY

*Secretary*, Dr. Robert L. Pinck, Long Island College Hospital, 340 Henry St., Brooklyn, N. Y. Meets first Thursday of each month, October through June.

##### BUFFALO RADIOLOGICAL SOCIETY

*Secretary*, Dr. Victor A. Panaro, 42 Burroughs Drive, Buffalo, N.Y. 14226. Meets second Monday evening each month, October to May inclusive.

##### CALIFORNIA RADIOLOGICAL SOCIETY

*Secretary*, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

##### CATAWBA VALLEY RADIOLOGICAL SOCIETY

*Secretary*, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. Meets every Tuesday, Dept. of Radiology, Valdeese General Hosp., Valdese, N. C., at 12:00 P.M.

##### CENTRAL NEW YORK RADIOLOGICAL SOCIETY

*Secretary-Treasurer*, Dr. Robert A. Bornhurst, State Univ. Hospital, 750 E. Adams St., Syracuse, N. Y. 13210. Meets first Monday each month, October through May.

##### CENTRAL OHIO RADIOLOGICAL SOCIETY

*Secretary*, Dr. Ollie E. Southard, 2787 Tudor Rd., Columbus, O. 43209. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

##### CENTRAL SOCIETY OF NUCLEAR MEDICINE

*Secretary*, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

##### CHICAGO ROENTGEN SOCIETY

*Secretary-Treasurer*, Dr. Fredric D. Lake, 2548 N. Lake-

\* Secretaries of societies are requested to send timely information promptly to the Editor.

- view Ave., Chicago, Ill. 60614. Meets second Thursday of each month, October to April, except December, at the Pick-Congress Hotel at 8:00 P.M.
- CLEVELAND RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Theodore J. Castele, 18869 Canyon Rd. Parkview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.
- COLORADO RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. George F. Wertz, 1801 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.
- CONNECTICUT VALLEY RADIOLOGIC SOCIETY**  
*Secretary*, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.
- DALLAS-FORT WORTH RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, John S. Alexander, 1217 W. Cannon, Ft. Worth, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:30 P.M.
- DETROIT ROENTGEN RAY AND RADIUM SOCIETY**  
*Secretary*, Dr. Robert L. Willis, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.
- EAST BAY RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Tom H. Piatt, 12 Camino Encinas, Orinda, Calif. 94563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.
- EAST TENNESSEE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. C. H. Kimball, 2200 Harris Circle, Cleveland, Tenn. Meets in January and September.
- EASTERN RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.
- FLORIDA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. John C. Jowett, Orlando, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.
- FLORIDA WEST COAST RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.
- GEORGIA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Al Rayle, Jr., 938 Peachtree St., N. E., Atlanta, Ga. Meets in spring and fall with Annual State Society Meeting.
- GREATER MIAMI RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Arthur R. Miller, North Miami General Hospital, 1701 N.E. 127th St., North Miami, Fla. Meets monthly, third Wednesday at 8:00 P.M., at Jackson Memorial Hospital, Miami, Fla.
- GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS**  
*Secretary-Treasurer*, John W. Fries, 3520 Chippewa St., St. Louis, Mo. 63118.
- HAWAII RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Donald Ikeda, Kuakini Hospital, 347 N. Kuakini St., Honolulu, Hawaii 96817. Meets third Monday of each month at 7:30 P.M.
- HOUSTON RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. William A. Vint, 1004 Seymour, Pasadena, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.
- IDaho STATE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.
- ILLINOIS RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.
- INDIANA ROENTGEN SOCIETY, INC.**  
*Secretary*, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.
- IOWA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.
- KANSAS RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.
- KENTUCKY CHAPTER, AMERICAN COLLEGE OF RADIOLOGY**  
*Secretary-Treasurer*, Dr. Robert H. Greenlaw, Dept. of Radiology, Univ. of Kentucky Med. Ctr., Lexington, Ky. Meets semiannually.
- KENTUCKY RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Joan R. Hale, 402 Heyburn Building, Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville, Ky.
- KINGS COUNTY RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.
- KNOXVILLE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.
- LONG ISLAND RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Samuel Gelband. Meets second Tuesday of the month in February, April, June, October and December.
- LOS ANGELES RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Norman Zheutlin, 8720 Beverly Blvd., Los Angeles, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.
- LOUISIANA TEXAS GULF COAST RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Texas 77701.
- MAINE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April.
- MARYLAND RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Henry Startzman, Medical Arts Building, Baltimore, Md.
- MEMPHIS ROENTGEN SOCIETY**  
*Secretary-Treasurer*, Dr. Vernon I. Smith, Jr., Suite 203, 1085 Madison Ave., Memphis, Tenn. 38104. Meets first Monday of each month at John Gaston Hospital.
- MIAMI VALLEY RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Indiana. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.
- MID-HUDSON RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.
- MILWAUKEE ROENTGEN RAY SOCIETY**  
*Secretary-Treasurer*, Dr. Donald P. Babbitt, 1700 W. Wisconsin Ave., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.
- MINNESOTA RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Edward A. Peterson, 572 Lowry Medical Arts Bldg., St. Paul, Minn. Meets twice annually, fall and winter.
- MISSISSIPPI RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.
- MISSOURI RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. M. Shoss, Cape Girardeau, Mo.
- MONTANA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.
- NEBRASKA STATE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.
- NEVADA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.



**NEW ENGLAND ROENTGEN RAY SOCIETY**

*Secretary*, Dr. Jack R. Dreyfuss, Zero Emerson Place, Boston, Mass. 02114. Meets third Friday of each month, October through May, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

**NEW HAMPSHIRE ROENTGEN RAY SOCIETY**

*Secretary*, Dr. Paul Y. Hassserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

**NEW MEXICO ASSOCIATION OF RADIOLOGISTS**

*Secretary-Treasurer*, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

**NEW MEXICO SOCIETY OF RADIOLOGISTS**

*Secretary*, Dr. William G. McPherson, Hobbs, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

**NEW YORK ROENTGEN SOCIETY**

*Secretary*, Dr. Arnold L. Bachman, Francis Delafield Hosp., 99 Fort Washington Ave., New York, N. Y. 10032. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Meeting: Waldorf Astoria Hotel, N. Y., April 27-29, 1967.

**NORTH CAROLINA RADIOLOGICAL SOCIETY**

*Secretary*, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

**NORTH DAKOTA RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Robert J. Olson, 1240 8th Ave., Williston, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

**NORTH FLORIDA RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

**NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

**NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY**

*Secretary-Treasurer*, Dr. John Turner, 1215-28th St., Sacramento, Calif. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

**NORTHWESTERN OHIO RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

**OHIO STATE RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Mortimer Lubert, Mt. Sinai Hospital, Cleveland, Ohio. Annual meeting to be announced.

**OKLAHOMA STATE RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Robert Sukman, 1200 N. Walker, Oklahoma City, Okla. Meets in January, May and October.

**ORANGE COUNTY RADIOLOGICAL SOCIETY**

*Secretary*, Dr. George W. Logan, 301 Newport Blvd., Newport Beach, Calif. Meets fourth Tuesday of every month at Orange County Medical Association Building.

**OREGON RADIOLOGICAL SOCIETY**

*Secretary-Treasurer*, Dr. Irving J. Horowitz, 2311 N.W. Northrup Str., Portland, Ore. 97210. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

**ORLEANS PARISH RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

**PACIFIC NORTHWEST RADIOLOGICAL SOCIETY**

*Secretary-Treasurer*, Dr. Willis Taylor, 1118 9th Ave., Seattle, Washington. Annual meeting to be announced.

**PENNSYLVANIA RADIOLOGICAL SOCIETY**

*Secretary*, Dr. T. Frederick Weiland, 619 Ridgeway Ave., Grove City, Pa. Annual meeting to be announced.

**PHILADELPHIA ROENTGEN RAY SOCIETY**

*Secretary*, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

**PITTSBURGH ROENTGEN SOCIETY**

*Secretary*, Dr. Robert N. Berk, 9100 Babcock Blvd., Pittsburgh, Pa. 15237. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

**RADIOLOGICAL SOCIETY OF CONNECTICUT, INC.**

*Secretary-Treasurer*, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held quarterly.

**RADIOLOGICAL SOCIETY OF GREATER CINCINNATI**

*Secretary*, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

**RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY**

*Secretary*, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

**RADIOLOGICAL SOCIETY OF KANSAS CITY**

*Secretary*, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

**RADIOLOGICAL SOCIETY OF LOUISIANA**

*Secretary*, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually, during Louisiana State Medical Society meeting and 6 months later.

**RADIOLOGICAL SOCIETY OF NEW JERSEY**

*Secretary*, Dr. John W. Marquis, 12 Hawthorne Ave., East Orange, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

**RADIOLOGICAL SOCIETY OF RHODE ISLAND**

*Secretary-Treasurer*, Dr. John M. Vesey, 1196 Elmwood Ave., Cranston, R. I.

**RADIOLOGICAL SOCIETY OF SOUTH DAKOTA**

*Secretary-Treasurer*, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

**RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA**

*Secretary-Treasurer*, Dr. Gerald M. McDonnell, U.C.L.A. Medical Center for Health Sciences, Los Angeles, Calif. 90024. Meets three times a year, usually October, February and May.

**RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK**

*Secretary-Treasurer*, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.

**REDWOOD EMPIRE RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

**RICHMOND COUNTY RADIOLOGICAL SOCIETY**

*Secretary*, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

**ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.**

*Secretary*, Dr. Irving B. Joffe, Rochester General Hospital, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

**ROCKY MOUNTAIN RADIOLOGICAL SOCIETY**

*Secretary-Treasurer*, Dr. Robert W. Lackey, 4200 E. Ninth Ave., Denver, Colo. Annual meeting to be announced.

**SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Hugh F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday of each month in Fort Sam Houston Officer's Club at 6:30 P.M.

**SAN DIEGO RADIOLOGICAL SOCIETY**

*President-Secretary*, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.

**SAN FRANCISCO RADIOLOGICAL SOCIETY**

*Secretary*, Dr. H. Joachim Burhenne, Children's Hospital and Adult Medical Center, 3700 California St., San Francisco, Calif. 94119. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco, Calif. 94118.

**SANTA CLARA COUNTY RADIOLOGICAL SOCIETY**

*Secretary*, Dr. Bill G. Karras. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

**SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION**

*Secretary*, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

**SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA**

*Secretary-Treasurer*, Dr. Gregory T. Henesy, 915 19th St., N. W., Washington, D. C. 20006. Meets at Medical Soci-

- ety Library, third Wednesday of January, March, May and October at 8:00 P.M.
- SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION**  
*Secretary*, Dr. Andrew F. Giesen, Jr., White-Wilson Clinic, Fort Walton Beach, Fla.
- SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION**  
*Secretary*, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.
- SHREVEPORT RADIOLOGICAL CLUB**  
*Secretary*, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.
- SOCIETY FOR PEDIATRIC RADIOLOGY**  
*Secretary*, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Washington Hilton Hotel, Washington, D. C., Sept. 25, 1967.
- SOCIETY OF NUCLEAR MEDICINE**  
*Secretary*, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.
- SOUTH BAY RADIOLOGICAL SOCIETY**  
*Secretary*, Northern Section: Dr. John H. Callaghan, 2900 Whipple Ave., Redwood City, Calif.; Southern Section: Dr. Carleton J. Wright, 2015 Clarman Way, San Jose, Calif. Meets second Wednesday of each month.
- SOUTH CAROLINA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.
- SOUTH DAKOTA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.
- SOUTHERN RADIOLOGICAL CONFERENCE**  
*Secretary-Treasurer*, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting to be announced.
- SOUTHWESTERN RADIOLOGICAL SOCIETY**  
*Secretary*, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.
- TENNESSEE RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Dr. E. K. Carter, Holston Valley Community Hosp., Kingsport, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.
- TEXAS RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Herman C. Schested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting to be announced.
- TRI-STATE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.
- UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING**  
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.
- UPPER PENINSULA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. A. Gonty, Menominee, Mich. Meets quarterly.
- UTAH STATE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Carlisle C. Smith, Salt Lake General Hospital, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.
- VERMONT RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. John R. Williams, 160 Allen St., Rutland, Vt.
- VIRGINIA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.
- WASHINGTON STATE RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.
- WEST VIRGINIA RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. George G. Green, Morgantown, W. V. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.
- WESTCHESTER COUNTY RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Arnold Gerson, Medical Arts Bldg., Mt. Vernon, N. Y. Meets on third Tuesday of January and October and on two other dates.
- WISCONSIN RADIOLOGICAL SOCIETY**  
*Secretary-Treasurer*, Harold F. Ibach, 2400 W. Villard Ave., Milwaukee, Wis. 53209. Meets twice a year, May and September.
- WYOMING RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. Ronald R. Lund, 240 W. 9th St., Casper, Wyo. Meets in fall with State Medical Society and in spring on call of President.
- CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA**  
**ASOCIACIÓN DE RADIOLOGOS DE CENTRO AMERICA Y PANAMÁ.** Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.  
*Secretary-General*, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.
- SOCIEDAD DE RADIOLOGÍA DE EL SALVADOR**  
*Secretary*, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador.
- SOCIEDAD DE RADIOLOGÍA DE GUATEMALA**  
*Secretary*, Dr. Carlos E. Escobar, 9a. Calle A o-o5, Zona 1, Guatemala.
- SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA CUBANA**  
*Secretary*, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.
- SOCIEDAD COSTARRICENSE DE RADIOLOGIA**  
*Secretary*, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.
- SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.**  
Coahuila No. 35, México 7, D. F.  
*Secretary-General*, Dr. Armando L. Rodríguez. Meets first Monday of each month.
- ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA**  
*Secretary*, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.
- SOCIEDAD RADIOLOGICA PANAMEÑA**  
*Secretary*, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.
- SOCIEDAD RADIOLOGICA DE PUERTO RICO**  
*Secretary*, Dr. José T. Medina, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.
- BRITISH COMMONWEALTH OF NATIONS**  
**ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC**  
*Secretary*, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.
- BRITISH INSTITUTE OF RADIOLOGY**  
*Honorary Secretary*, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May. Annual meeting: Central Hall and Caxton Hall, Westminster, London, S. W. 1., England, April 26-28, 1967.
- CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.**  
*Honorary Secretary-Treasurer*, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting to be announced.
- EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY**  
*Secretary*, Dr. B. V. Evans, 105 Northgate Bldg., Alberta, Canada. Meets second Tuesday of each month in various Edmonton Hospitals.
- FACULTY OF RADIOLOGISTS**  
*Honorary Secretary*, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting Guy's Hospital, London, June 23-24, 1967.
- FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND**  
*Registrar*, Dr. H. O'Flanagan, F.R.C.P.L., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.



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*Secretary*, Juan Gomez Lopez, Villanueva, 11, Madrid 1. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid. Annual general meeting to be announced.

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# ABSTRACTS OF RADIOLOGICAL LITERATURE

## INDEX TO ABSTRACTS

### ROENTGEN DIAGNOSIS

#### HEAD

- McRAE, D. L., and MAKOW, M.: Horizontal laminography of the head with ultra-sound. 1014
- AKERMAN, M., and GUIOT, G.: The diagnosis of intracranial space-occupying lesions by association of gamma-encephalography and echo-encephalography. 1014
- POSNIKOFF, J., and SARGENT, E. N.: Multiple bilateral intracranial intracavernous and intradural carotid aneurysms: a compendium of classical findings—case report. 1014

#### NECK AND CHEST

- KEGGI, K. J., GRANGER, D. P., and SOUTHWICK, W. O.: Vertebral artery insufficiency secondary to trauma and osteoarthritis of the cervical spine. 1014
- DE VILLIERS, J. C.: A brachiocephalic vascular syndrome associated with cervical rib. 1015
- FELSON, B.: Disseminated interstitial diseases of the lung. 1015
- DEES, S. C., and SPOCK, A.: Right middle lobe syndrome in children. 1016
- CONSTANTINE, H., DESFORGES, G., and GAENSLE, E. A.: Noninfectious necrotizing granulomatosis of the lung: Wegener's syndrome. 1016
- FLAVELL, G.: Lung abscess. 1017
- MACKENZIE, C. J. G.: Non-tuberculous chest disease found in a mass x-ray survey in Vancouver, B. C. 1017
- RICHARDSON, P. *et al.*: Pulmonary oil deposition in patients subjected to lymphography: detection by thoracic photoscan and sputum examination. 1017
- SIRTORI, C.: Cytodiagnosis and pathogenesis of lung tumors and experiments with influenza virus: optical and electron microscopic studies. 1018
- LEGER, L. *et al.*: Radiologic examination of the cardio-esophageal junction in profile (in upright position, with the trunk bent and in decubitus position). 1018

#### ABDOMEN

- KAHN, I. J., JEFFRIES, G. H., and SLEISINGER, M. H.: Malabsorption in intestinal scleroderma: correction by antibiotics. 1018

- MATHISEN, W., and HOLTA, A. L.: Idiopathic retroperitoneal fibrosis. 1019
- GUIEN, C. *et al.*: Tomographic exploration of abdominal viscera by combined pneumoperitoneum and retroperitoneum. 1019
- PIETRI, H. *et al.*: Tomography of the liver and pancreas with combined pneumoperitoneum and retroperitoneum. 1019
- TESCHENDORF, W., and SCHIRMEISEN, P.: Oral cholangiography in combination with gastrointestinal examination. 1020
- BUCHTALA, V.: Peroral cholangio-cholecystography with SH 771: results and side effects in 766 examinations. 1020

### RADIATION THERAPY

- JEFFERSON, A.: Symposium on pituitary tumours: I. Clinical management and the results of treatment of the chromophobe pituitary adenoma. 1020
- LEWTAS, N. A.: Symposium on pituitary tumours: II. Radiology in diagnosis and management. 1021
- EMMANUEL, I. G.: Symposium on pituitary tumours: III. Historical aspects of radiotherapy, present treatment technique and results. 1021
- WOLMAN, L.: Symposium on pituitary tumours: IV. Unusual pathological features. 1022
- ROBBINS, G. F. *et al.*: An evaluation of post-operative prophylactic radiation therapy in breast cancer. 1022
- BUSCHKE, F.: Some reflections of the treatment and prognosis of Hodgkin's disease. 1022
- CONRADY, J. *et al.*: Pelvic angiography and lymphangiography in the evaluation of the patient with carcinoma of the cervix. 1023
- LANG, E. K. *et al.*: The accuracy of preoperative staging of bladder tumors by arteriography: a 5-year study. 1023
- RUDD, K. D. *et al.*: Radium source appliance for treatment of nasopharyngeal cancer. 1024
- BABINI, L., and SASSI, P.: Perforations and fistulae in the course of radiotherapy of malignant tumors of the esophagus. 1024
- ZUPPINGER, A.: The importance of biology in radiotherapy. 1024



- MELLINS, F. *et al.*: The effect of high energy laser pulses on the primate liver..... 1025  
 CADE, S.: Iatrogenics..... 1025  
 RIGBY, C. C.: The pathology of rhabdomyosarcoma in children..... 1026

## RADIOISOTOPES

- LOKEN, M. K. *et al.*: Mercury-197 and mercury-203 chlormerodrin for evaluation of brain lesions using a rectilinear scanner and scintillation camera..... 1026  
 SCHMUKLER, M., and WORKMAN, J. B.: The reliability of scintillation scanning for detection of intracranial lesions..... 1026  
 GREENBERG, W. V.: Thyroidal <sup>131</sup>I turnover in hypothyroidism: correlation with thyrotropin responsiveness..... 1026  
 MORRIS, J. G. *et al.*: Lung scanning—technique and some diagnostic uses..... 1027  
 TAPLIN, G. V., POE, N. D., and GREENBERG, A.: Lung scanning following radioaerosol inhalation..... 1027  
 SHEHADI, W. H.: Practical applications of liver scanning..... 1028  
 CAVALIERI, R. R., SCOTT, K. G., and SAIRENJI, E.: Selenite (<sup>74</sup>Se) as a tumor-localizing agent in man..... 1028  
 DENARDO, G. L., and VOLPE, J. A.: Detection of bone lesions with the strontium-85 scintiscan..... 1029  
 GOTTSCHALK, A. *et al.*: Quantification of the

- respiratory motion artifact in radioisotope scanning with the rectilinear focused collimator scanner and the gamma scintillation camera..... 1029  
 DAVIS, T. P., and MARTONE, R. J.: The hybrid radioisotope scanner..... 1029  
 HORWITZ, N. H., LOFSTROM, J. E., and FORSAITH, A. L.: A comparison of clinical results obtained with a spark-imaging camera and a conventional scintillation scanner..... 1029  
 DUGGAN, H. E., and WEIJER, D. L.: Polycythemia rubra vera and radioactive phosphorus—50 patients..... 1030

## CHEMOTHERAPY

- FRECKMAN, H. A. *et al.*: Chemotherapy for lung cancer in intra-aortic infusion..... 1030

## MISCELLANEOUS

- MACKEY, J. S., and ROSS, R. C.: Hepatoma induced by thorium dioxide (thorotrast)... 1030  
 BIZZOZERO, O. J., JR., JOHNSON, K. G., and CIOCCO, A.: Radiation-related leukemia in Hiroshima and Nagasaki, 1946–1964: I. Distribution, incidence and appearance time... 1031  
 CONARD, R. A., RALL, R. E., and SUTOW, W. W.: Thyroid nodules as a late sequela of radioactive fallout in a Marshall island population exposed in 1954..... 1031  
 WARD, G. M., JOHNSON, J. E., and WILSON, D. W.: Deposition of fallout cesium 137 on forage and transfer to milk..... 1031



## ROENTGEN DIAGNOSIS

## HEAD

McRAE, D. L., and MAKOW, M. Horizontal laminography of the head with ultra-sound. *J. Canad. A. Radiologists*, June, 1966, 17, 75-80. (From: Department of Radiology, Montreal Neurological Institute, Montreal, Quebec, and National Research Council, Division of Applied Physics, Ottawa, Ontario, Canada.)

The usefulness of laminography in diagnostic radiology is widely accepted. This principle of controlled plane scanning has been adapted to the newer technique of ultrasound cerebral scanning, and initial clinical trials in both normal and abnormal patients are reported.

The technique requires immersion of that horizontal plane of the skull to be scanned in water. A transducer then scans the head by traveling about the circumference of the immersion bath. The plane of interest may be selected or changed at will.

Thirty-three patients have been evaluated by using this technique. Control studies were performed on the same patients using cerebral arteriography and/or pneumoencephalography. In 21 of these no organic pathology was detected. Ultrasound laminography correctly located the third ventricle in 15 of these patients and incorrectly located it in 3. In 6, the lateral ventricles were accurately defined, and, in addition, the brain stem and insula were located spatially in several patients. Twelve of the patients were found to have gross abnormalities of the brain including tumors, postoperative defects, atrophy, and Wernicke's encephalopathy. These patients had 14 scans; 13 revealed the third ventricle and in 9 it was correctly located.

The technique, however, failed to detect a brain abscess, a subdural collection of fluid, and a calcified aneurysm.—*Frederick R. Cushing, M.D.*

AKERMAN, M., and GUIOT, G. Le diagnostic des processus expansifs intra-craniens par l'association de la gamma et de l'écho-encéphalographie (étude statistique de 650 cas). (The diagnosis of intracranial space-occupying lesions by association of gamma-encephalography and echo-encephalography.) *Ann. de radiol.*, 1965, 8, 851-879. (From: Unité de Recherches de Neurophysiologie chirurgicale I.N.S.E.R.M., Service de Neuro-chirurgie et Laboratoire des Isotopes de l'Hôpital Foch, Suresnes, France.)

The value of gamma-encephalography is already well known. It is interesting to note that the authors report results nearly as favorable for the lesions of the posterior fossa as for the supratentorial lesions.

The results of echo-encephalography depend essentially on the location of mass lesions and is, therefore, more efficacious in lesions of the cerebral hemispheres. This method has the advantage of simplicity and repetitiveness with which it may be applied to cases of extreme urgency.

When both studies are performed, echo-encephalography reinforces the opinion of a unilateral lesion which may have been diagnosed by gamma-encephalography. Also, it permits the discovery of displacements which may escape gamma-encephalography, and may aid in interpreting certain areas of hyperactivity which are not caused by tumors. Examples include postoperative softening or scar formation.—*Charles M. Nice, Jr., M.D., Ph.D.*

POSNIKOFF, JACK, and SARGENT, E. NICHOLAS.

Multiple bilateral intracranial intracavernous and intradural carotid aneurysms: a compendium of classical findings—case report. *Bull. Los Angeles Neurologic. Soc.*, April, 1966, 31, 51-62. (From: Department of Neurosurgery, and Radiology Services, Orange County General Hospital, Orange, Calif.)

A classic case of bilateral intracranial carotid aneurysms in a 77 year old woman is reported. The plain roentgenologic findings included a curvilinear calcification well above the left side of the sella turcica, enlargement of the entire sella with a sharp double contour to the floor, demineralization and erosion of the dorsum sellae, and loss of the posterior clinoid processes. Both anterior clinoids were also eroded. The patient's primary clinical symptoms were relative to a right corneal ulceration following loss of vision in that eye. She was a controlled hypertensive patient for 8 years.

Angiography demonstrated bilateral saccular intracavernous, extradural, parasellar aneurysms. These were surmounted by intradural, supraclinoid, carotid aneurysmal dilatations. In this case, the complete loss of the dorsum sellae was differentiated from the physiologic senile demineralization in which the cortical outline remnant is usually preserved. Atrophy of the homolateral optic foramen can occur, and enlargement of the foramen lacerum may be seen in the basilar views. Large aneurysms can cause destruction of the floor of the middle fossa and changes in the superior orbital fissure.—*Arch H. Hall, M.D.*

## NECK AND CHEST

KEGGI, KRISTAPS J., GRANGER, DONALD P., and SOUTHWICK, WAYNE O. Vertebral artery insufficiency secondary to trauma and osteoarthritis of the cervical spine. *Yale J. Biol. & Med.*, April, 1966, 38, 471-478. (From: Department of Surgery, Section of Ortho-



pedic Surgery, Yale University School of Medicine, and the West Haven Veterans Administration Hospital, New Haven, Conn.)

Obstruction of the vertebral arteries can lead to sudden death, extensive spinal cord destruction, insufficiency of the brain stem, impaired cerebellar function, damage to the occipital cortex and may be responsible for certain forms of cerebral palsy.

A case of post-traumatic vertebral artery insufficiency is reported in which the diagnosis was not made until approximately 1 year after the original trauma. Vascular studies showed compression of the left vertebral artery by an osteophyte, while the artery on the right side was completely thrombosed. Surgical decompression and anterior interbody fusion gave the patient almost complete relief from symptoms.

It is wise to bear in mind the possibility of vertebral artery occlusion or thrombosis in any case of cervical trauma. One should even be more alert for the condition if there are complicating factors of arteriosclerosis, hypertrophic osteoarthropathy, severe muscle spasm, tumors, or congenital anomalies.

In the case reported, subluxation, muscle spasm, and associated soft tissue trauma apparently caused the right vertebral artery thrombosis. The gradual post-traumatic osteophyte formation subsequently impaired blood flow in the remaining left vertebral artery which necessitated surgical intervention.—*Arch H. Hall, M.D.*

DE VILLIERS, J. C. A brachiocephalic vascular syndrome associated with cervical rib. *Brit. M. J.*, July 16, 1966, 2, 140-143. (Address: Department of Neurosurgery, Pennsylvania Hospital, Philadelphia, Pa.)

The author presents a case of cerebral hemiplegia caused by emboli arising from a thrombus in the right subclavian artery incident to vascular occlusion from a cervical rib. This is an extremely rare condition and only 8 other cases have been reported in the world literature. Symonds in 1927 first described the pathophysiology of this lesion. He suggested that a mural thrombus occurred in the subclavian artery as a result of local damage by the cervical rib. This thrombus extended backward to the point of junction with the right common carotid artery. A portion of the thrombus could then break off into the common carotid artery. The condition can only occur on the right side, because only on the right do the subclavian and carotid arteries have a common origin from the innominate artery.

The present case did not show evidence of such a massive thrombosis. No more than a small friable clot was found in the subclavian artery distal to the cervical rib. The author suggests that with certain

positions of the arm in the presence of a cervical rib, probably only tolerated during sleep, a complete occlusion of the subclavian artery could occur. Marked turbulence might then ensue in the proximal part of the vessel giving rise to small emboli.

In all cases of this condition previously described, symptoms of vascular insufficiency in the right arm preceded the cerebrovascular accident by a period varying from 3 months to 10 years.

The author concludes with a plea for early surgical treatment of the vascular changes often associated with cervical ribs in an effort to prevent the possible cerebral complications.—*Kenneth M. Nowicki, M.D.*

FELSON, BENJAMIN. Disseminated interstitial diseases of the lung. *Ann. de radiol.*, 1966, 9, 325-345. (From: University of Cincinnati College of Medicine and Medical Center, and Cincinnati Veterans Administration Hospital, Cincinnati, Ohio.)

The author discusses in depth the pathophysiology and radiographic features of the myriad of disease entities that are known to cause diffuse interstitial fibrosis of the lung. He begins by pointing out that many patients with severe respiratory symptoms who are proven to have the microscopic findings of an extensive interstitial disorder have normal chest roentgenograms. This is because the individual nodules or infiltrates are so small or ill-defined that their detectability falls below the level of resolution of the roentgen method. Many interstitial diseases, such as viral pneumonia and interstitial edema, do become evident, however, because of the rapid extension of the process into the alveolar spaces. Interstitial disease usually takes at least a week after the onset of symptoms, and sometimes longer, to become visible on standard chest roentgenograms. The fine nodular lesions of interstitial granulomatosis usually become visible only after they have reached 2-3 mm. in diameter. Miliary shadows actually represent summation images of superimposed nodules. Alveolar lesions may be nodular but are never as small or discrete as interstitial nodules. They usually measure over 0.5 cm. in diameter and tend to coalesce. The interstitial nodules of hematogenous metastases vary in size, are sharper, more rounded, less numerous than granulomatous nodules and hardly ever coalesce.

Another type of interstitial involvement is interstitial edema. Here the chest roentgenogram may be normal or show transient Kerley's A and C lines, which represent dilated lymphatic vessels, as opposed to B lines which represent swelling of the interlobular septa. Once pulmonary densities are seen, the edema is already predominantly alveolar.

One of the characteristic features of interstitial fibrosis is "honeycombing." This is due to an obstructive emphysema of the affected portion of the lung. Air is forced into this area via the valve-like

mechanism at the pores of Kohn in the adjacent normally ventilated alveoli. These air filled sacs have a diameter of from 1-10 mm., with the smaller sizes predominating. A patchy distribution is the most commonly seen type. The author believes that this is the only dependable sign of interstitial fibrosis.

A discussion of "honeycombing" and the disease entities causing diffuse interstitial fibrosis produced the following points of interest: Spontaneous pneumothorax was found to be a common feature of interstitial fibrosis, particularly in the presence of "honeycombing." Also, "honeycombing" may be indistinguishable from the nodulation of interstitial granulomatosis or cystic bronchiectasis. It is far easier to differentiate "honeycombing" from radiolucencies occasionally seen in alveolar infiltration, or the small cavities visible in pulmonary infiltrates associated with tuberculosis or fungus disease. Among the disease states capable of causing diffuse interstitial pulmonary fibrosis, the author lists the idiopathic form (Hamman-Rich syndrome); sarcoidosis; histiocytosis X, particularly eosinophilic granuloma; scleroderma; rheumatoid arthritis; pneumoconiosis; and chronic lipid pneumonia. Tuberculous sclerosis and muscular cirrhosis of the lungs give a roentgen picture similar to that of diffuse interstitial fibrosis; however, in these cases the interstitium consists predominantly of smooth muscle. In the newborn infant one may observe alike radiographic features caused by congenital adenomatoid malformation, a diffuse hamartomatous anomaly which is always unilateral, and the pulmonary dysmaturity syndrome (Wilson-Mikity disease) which regresses spontaneously in 50 per cent of the cases. Various rare conditions such as desquamative interstitial pneumonia, farmer's lung, inhalation of mercury fumes, streptomycin treated tuberculous bronchopneumonia, mucoviscidosis, idiopathic pulmonary hemosiderosis, and chronic mitral valve disease are also mentioned.

The author concludes by pointing out that "honeycombing" is often a recognizable roentgen feature and nearly always indicates interstitial fibrosis. The differential diagnosis of the causes of the honeycomb lung cannot be based on the radiographic picture alone. All available information must be utilized. As a general rule, the more frequent possibilities should always be considered first. Despite all leads, however, pulmonary biopsy is still indicated in most situations.

Twenty roentgenographic reproductions and 7 photomicrographs are included in this article.—*Kenneth M. Nowicki, M.D.*

DEES, SUSAN C., and SPOCK, ALEXANDER. Right middle lobe syndrome in children. *J.A.M.A.*, July 4, 1966, 197, 8-14. (From: Department of Pediatrics, Duke University Medical Center, Durham, N. C.)

The authors report a series of 30 children with right

middle lobe syndrome who had persistent cough and intermittent asthma-like wheezing. Recurrent pneumonia or bronchitis localized to the middle lobe were constant findings confirmed by roentgenologic examination. The symptoms differ in several respects from those reported for adults with this problem in whom cough, fever, purulent sputum and hemoptysis are primary features.

In this series, the onset of the symptoms was always before the age of 6 years and in over half of the cases, by 2 years of age. In 21 children, however, there was an average lag of 4 years between the initial symptoms and the diagnosis. The standard laboratory tests were not specific but an elevation of the hematocrit was observed in 25 of the 30, 3 were found to have dysgammaglobulinemia, and nasal eosinophilia was present in 23 of 25 studied. Leukocytosis and elevated ESR were constant findings.

All of the children had roentgenographic examinations including posteroanterior and right latero-chest views. In 20 cases, classic atelectasis of the right middle lobe was present. The findings in the other 10 were suggestive of atelectasis or infection of the right middle lobe with specific features including a blurred right heart border in 3 cases, decreased right middle lobe volume in 1, increased right middle lobe density in 2, and increased bronchovascular markings in 4. In this latter group of 10 cases with equivocal findings, bronchograms showed some abnormality in all 6 cases that were studied. Abnormal sinus roentgenograms were found in 19 of 24 cases studied.

Bronchoscopy was not as useful as a diagnostic test since only 7 of 17 studies showed any endobronchial changes, but often was mandatory to rule out foreign body, mucus plugs or endobronchial disease.

The authors feel that the right middle lobe syndrome is frequently seen in allergic children but unrecognized as such and the prognosis for recovery and the best results from surgery are directly related to early diagnosis and treatment.—*James R. Knapp, M.D.*

CONSTANTINE, H., DESFORGES, G., and GAENSLER, E. A. Noninfectious necrotizing granulomatosis of the lung: Wegener's syndrome. *Med. thorac.*, 1966, 23, 115-126. (From: Thoracic Services, Boston University School of Medicine, Boston, Mass.)

The case of a 40 year old female with noninfectious necrotizing granulomatosis of the lung is reported. She had a long history of arthritis and drug sensitivity and presented with signs and symptoms of an upper respiratory infection.

A chest roentgenogram showed a large solitary mass in the right upper lobe. A neoplasm was suspected and a lobectomy was performed. The histologic examination revealed noninfectious necrotizing granulomatosis.

Roentgenographic studies of the joints, sinuses and



kidneys showed no significant abnormalities. She is alive and well 1 year later.

A review of the literature is presented.—*John H. Harris, M.D.*

FLAVELL, GEOFFREY. Lung abscess. *Brit. M. J.*, April 23, 1966, 1, 1032-1036. (Address: Surgeon, Department of Thoracic Surgery, The London Hospital, London, England.)

Generally speaking, suppurating cavities in the lung require isolation of the specific etiologic agents and aggressive therapy. All patients must be bronchoscoped, either for diagnostic or therapeutic purposes, but not until the sputum has been examined for neoplastic cells. Roentgenologic examination, and especially tomography, aid in establishing the diagnosis, with periodic re-examination necessary to follow the course of the disease. If an air fluid level is present within the abscess, it communicates with a bronchus.

Abscesses associated with malignancies represent either necrosis and cavitation of a primary squamous cell carcinoma, or reflect a pyogenic abscess either distal to a partially obstructing neoplasm, or in a more dependent segment incident to infected spill-over from the neoplasm. In males over 50 years of age, these are by far the most common abscesses.

Aspiration abscesses usually are associated with gross dental sepsis and suppression of the cough reflex. Obstructive disease of the esophagus may also result in over-spill and aspiration of food during sleep. The possibility of alcoholism must also be borne in mind. The site of the abscess in the lung is determined by the posture of the patient at the moment of inhalation. If he is lying on his back, the septic debris are more likely to go down the right main bronchus, and the first dependent bronchial orifice on its floor is that leading to the apical segment of the right lower lobe. This, in fact, is the most common location of an aspiration abscess.

Infected pulmonary emboli and septic emboli from a general pyemia also may cause lung abscesses. Secondary infections of congenital or acquired lung cysts, dermoids, or sequestered segments are uncommon causes of lung abscesses, but should be considered if cavitation persists in a lung after appropriate treatment.

Of the specific organisms, *Staphylococcus aureus* is perhaps most common in infancy. This process frequently extends to the pleural space to produce an empyema. Characteristic peripheral thin-walled abscess-sacs rapidly appear, and may coalesce to form one large abscess cavity or pseudocyst. Needle aspiration of the pus in the pleural cavity is felt to be just as effective as tube drainage. Friedländer's bacillus is most common in elderly debilitated subjects and is characterized by large abscess cavities occupying the whole of one lobe and accompanied by massive sloughing of the necrotic lung. Actinomycosis is now rare but is recognized by its disregard for

tissue barriers and discharging pus through sinuses containing typical "sulphur granules." Amebic abscesses in the lungs are rare. The differential diagnosis of chronic pulmonary cavitation should always include tuberculosis.

Failure of the lung abscess to respond to aggressive therapy indicates either chronicity, a secondarily infected benign structure, or an underlying malignancy. For any of these, prompt surgical resection is indicated.—*Donald M. Monson, M.D.*

MACKENZIE, C. J. G. Non-tuberculous chest disease found in a mass x-ray survey in Vancouver, B. C. *Canad. M. A. J.*, June 11, 1966, 94, 1257-1261. (From: Department of Preventive Medicine, Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada.)

The wisdom of continuing chest x-ray surveys has been questioned in recent years because of the declining incidence of tuberculosis. In Vancouver, B.C., 115 new cases were detected in a 1964 survey of 219,000 individuals.

In the same survey, significant non-tuberculous chest disease was disclosed in 929 patients, representing previously unsuspected disease in about one-third of instances.

The type of lesion and required follow-up are analyzed in some detail; approximately one-quarter of the cases falling into an urgent follow-up category with lesions such as masses, effusion, aneurysms and infiltrates. Thirty-nine cases of bronchogenic carcinoma are included.

The need for a pre-arranged follow-up system is emphasized by the failure of 26 per cent of the patients, classified as having significant disease, to contact their physician.—*Rosalind H. Troupin, M.D.*

RICHARDSON, P., CROSBY, E. H., BEAN, H. A., and DEXTER, D. Pulmonary oil deposition in patients subjected to lymphography: detection by thoracic photoscan and sputum examination. *Canad. M. A. J.*, May 21, 1966, 94, 1086-1091. (From: Allan Blair Memorial Clinic and Regina Grey Nuns' Hospital, Regina, Saskatchewan, Canada.)

Reports of pulmonary oil embolism following lymphography have varied from 17 to 55 per cent by radiographic detection.

The authors, during clinical trials of intralymphatic therapy with radioiodinated ethiodized oil for malignant disease of lymph nodes, found significant radioactive material in the lungs in 5 consecutive cases done (thoracic scans). Radioactivity was present in the sputa of 2 of these cases. No cases showed radiographic evidence of emboli.

Conservative techniques were used during injection. Despite the limitation of volume of oil injected, monitoring of the infusion, and absence of radio-

graphic evidence of contrast material in the lungs, significant pulmonary deposition of oil appears to be inevitable following lymphography.—*George A. Miller, M.D.*

SIRTORI, C. Cytodiagnosis and pathogenesis of lung tumors and experiments with influenza virus: optical and electron microscopic studies. *Panminerva Med.*, April, 1966, 8, 103-110. (From: Morbid Anatomy Division, National Cancer Institute, Milan, Italy.)

Cytodiagnosis has now emerged from its infancy as demonstrated by the achievement of diagnostic effort acclaimed for the diagnosis of carcinoma of the cervix. Less enthusiasm exists, however, in the diagnosis of carcinoma of the lung.

The author describes a technique for improvement over the regular Papanicolaou smear by the utilization of carefully collected sputum specimens subsequently imbedded in paraffin, sectioned and stained with hematoxylin and eosin. The specimens are collected directly into denatured alcohol and subsequently subjected to further fixation in Dubosq-Brazil fluid. He has examined 3,433 specimens with 1,045 positive results, 1,963 negative results, 172 doubtful results and 253 inconclusive results owing to insufficient salivary material. It is his opinion that a correct diagnosis can be made in 93 per cent of the cases.

The author stresses the importance of the method used to collect the sputum, elaborates on the diagnostic difficulties concerning phantom cells, India ink cells, and the possibility of roentgenologically inapparent tumors with a positive cytology.

Paraffin fixation of sputum, in his opinion, is preferred to Papanicolaou type smears since it presents no problem in a well equipped morbid anatomy laboratory and presents the examiner with cells stained by usual techniques familiar to the pathologist.

In the author's laboratory, investigations with influenza virus have been carried on in which he has shown that the virus forms especially in the cells responsible for the alveolar tensioactive substance and, therefore, for the elasticity of the lung. Lesions of these cells favor the accumulation of inhaled carcinogens and for this reason he suggests that influenza virus can be considered as a co-factor in the etiology of lung cancer.—*Luther W. Brady, M.D.*

LEGER, LUCIEN, LEDOUX-LEBARD, GUY, KANOUI, FERNAND, and PICARD, CLAUDE. (Paris, France.) L'examen radiologique de la région cardio-oesophago-tubéreuse de profil (en position debout, tronc fléchi et en position couchée). (Radiologic examination of the cardio-esophageal junction in profile [in upright position, with the trunk bent and

in decubitus position].) *Presse méd.*, Suppl., June, 1966, 74, 31-34.

The difficulties in roentgenologic exploration of the gastric fundus, the cardia, and esophageo-cardiac junction, and of making an accurate diagnosis, both by roentgenologic examination as well as by esophagoscopy, and gastroscopy, are well known. In the examination of this area the authors stress the importance of the lateral profile projection in both the upright and the horizontal position.

The examination is performed in the upright position, with the patient's body bent forward as much as possible. Orally administered barium clearly defines the distal esophagus, and with air in the stomach, gives a double contrast study of the cardia and fundus. This technique is quite helpful in the demonstration of hiatal hernia and particularly of gastric esophageal reflux.

Studies of the same area with the patient in the horizontal position may be done in both the prone and supine position. In the prone position, the gas bubble rises towards the spine, giving a double contrast which is particularly helpful in a study of the mucosa and in the detection of ulceration and tumor formation in the posterior aspect of the fundus. With the patient in the supine position, the opposite aspect of the fundus is more readily visible.

With the patient in the horizontal position, a grid cassette is placed vertically on the table against the side of the patient, and a horizontal beam at right angle to the cassette is obtained by placing the tube on the contralateral side.

Lesions of the cardia are readily outlined, and varices become more apparent. In the examination of the gastrointestinal tract, the authors routinely obtain a lateral profile roentgenogram of the fundus with the patient in both the prone and supine position.

(During the past 20 years, this reviewer has used, in selected cases, the horizontal profile projection in both the decubitus and supine position. The clarity of detail of the fundus and the esophageo-gastric junction, particularly in the detection of ulcers and tumors, which would otherwise be obscured, is most rewarding.)

Fifteen illustrations and reproductions of roentgenograms accompany this informative article.—*William H. Shehadi, M.D.*

#### ABDOMEN

KAHN, IVAN J., JEFFRIES, GRAHAM H., and SLEISINGER, MARVIN H. Malabsorption in intestinal scleroderma: correction by antibiotics. *New England J. Med.*, June 16, 1966, 274, 1339-1344. (Address: Dr. Jeffries, 525 East 68th Street, New York, N. Y.)

Correction of malabsorption in intestinal scleroderma in 3 out of 4 patients treated with a broad-



spectrum antibiotic is reported. The etiology of malabsorption in patients with intestinal scleroderma has never been satisfactorily explained, and, among other things, the possibility of overgrowth of bacteria in atonic loops of bowel has been considered.

Each of the patients treated had had evidence of disseminated scleroderma for many years before the onset of intestinal symptoms. The presence of malabsorption was proved by the use of sensitive absorption tests, including D-xylose excretion and quantitative measurement of fecal fat excretion by the method of van de Kamer *et al.*

Involvement of the small intestine in scleroderma is common enough, but true malabsorption has been reported infrequently. Bacterial proliferation in the upper small bowel was confirmed by culture of aspirated duodenal contents.

Two of the patients studied have been maintained on long term antibiotic therapy with satisfactory well-being and weight gain. The development of recurrent symptoms was ascribed to bacterial resistance requiring a change in the antibiotic being used. Tetracycline therapy was most dramatic causing a cessation of abdominal distention, vomiting and diarrhea within 12 hours. Oxytetracycline and ampicillin were successfully used to overcome symptoms recurring after bacterial resistance to tetracycline developed. Repeated duodenal cultures could not be obtained to prove presence of antibiotic-resistant micro-organisms, however.

This therapy in no way influences the basic pathologic process in scleroderma and presumably there is no change in the usual radiologic abnormalities typical of intestinal disease.—*Richard E. Kinzer, M.D.*

MATHISEN, WILLY, and HOLTA, ANNE LISE. Idiopathic retroperitoneal fibrosis. *Surg., Gynec. & Obst.*, June, 1966, 122, 1278-1282. (From: Division of Urology, University Hospital, Rikshospitalet, Oslo, Norway.)

There is a close similarity between the microscopic appearance of the tissues in retroperitoneal fibrosis and chronic lymphedema of the lower extremities. It is believed that the pathogenesis is the same. The process begins with a lymphangitis, probably of viral origin. This results in lymph stasis and dilatation of the lymphatic vessels. Protein rich fluid containing lymphocytes escapes into the extracellular space in the retroperitoneal area. The proteins are precipitated and collagenous tissue is formed following the invasion of fibroblasts. Superfluous water is reabsorbed causing the fibrous tissue to retract toward the midline so that structures included in the process are compressed and displaced.

Nine patients with surgically verified retroperitoneal fibrosis were studied. Four patients with sclerosing mediastinitis exhibited the same pathologic appearance.—*David Morse, M.D.*

GUIEN, C., PIETRI, H., SARLES, H., and LEGRÉ, J. (Marseille, France.) L'exploration tomographique des viscères abdominaux par pneumopéritoine et rétropneumopéritoine associés. (Tomographic exploration of abdominal viscera by combined pneumoperitoneum and rétropneumoperitoneum.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1966, 47, 321-324.

There are 3 modalities of tomography: frontal, sagittal and transverse axial. There are 3 methods of enhancing contrast by gas: pneumoperitoneum, rétropneumoperitoneum and gastric insufflation. The originality of the authors' method consists in using the proper combination of these approaches with small quantities of gas, so that each organ is dissociated from its bed by suitable insufflations and positioning, with tomography furnishing virtually panoramic views.

The failure to separate organs as expected indicates that they are adherent or invaded. The liver can be evaluated as to volume, compensatory hypertrophies or other affections. No other method allows determination of splenic volume with such precision. Frontal tomography alone is adequate to delineate the pancreas. Kidneys, suprarenals and deep lymph node masses are well brought out, and, finally, the great vessels are completely visualized.

The indications for this complementary approach fall into two groups: exploration of a specific organ for study of its volume, morphology and relationships to adjacent tissues, or search for a possible abdominal lesion, with recognition of inflammatory or metastatic invasions.—*Frank A. Riebel, M.D.*

PIETRI, H., GUIEN, CL., SARLES, J.-CL., ASSADOURIAN, R., SARLES, H., and LEGRÉ, J. Tomographie du foie et du pancréas sous pneumopéritoine et rétropneumopéritoine associés. (Tomography of the liver and pancreas with combined pneumoperitoneum and rétropneumoperitoneum.) *Ann. de radiol.*, 1965, 8, 765-784. (From: Unité de Recherche de Pathologie Digestive, Hôpital Sainte-Marguerite, Marseille, and Chaire d'Electroradiologie de la Faculté de Médecine de Marseille, Hôpital de la Timone, Marseille, France.)

A systematic combination of pneumoperitoneum and rétropneumoperitoneum has proved quite useful in examining the liver. Surface irregularities caused by regeneration nodules of cirrhosis, tumors or metastases are clearly shown. The inferior vena cava can be observed in its intrahepatic tract. Calcifications in the right hypochondrium can be accurately localized. Hypertrophy of the caudate lobe is shown extremely well.

From frontal and transverse tomograms the shape of the liver can be determined and a model of the organ constructed. From this model the volume of the liver can be deduced.

In frontal tomograms, the outline of the body and tail of the pancreas is clearly defined. If the head of the pancreas is to be examined, an air contrast injection in the duodenum may be useful. The liver and pancreas are clearly seen in their relation to other organs and anatomic elements.—*Charles M. Nice, Jr., M.D., Ph.D.*

TESCHENDORF, W., and SCHIRMEISEN, P. Orale Cholangiographie in Verbindung mit der Magen-Darm-Untersuchung. (Oral cholangiography in combination with gastrointestinal examination.) *Röntgen Blätter*, May, 1966, 19, 248-257. (Address: Strahleninstitut der AOK Köln, Machabäerstrasse 19-27, 5 Köln, Germany.)

Oral cholangiography with available contrast media has only been partly successful thus far. A new contrast medium—SH 771 (Schering)—, however, proved superior for visualization of the bile ducts. Its concentration in the secreted bile is twice as high as that of other media. The gallbladder could readily be visualized 2 to 4 hours after the ingestion of 3 gm. of SH 771 in 92 of 100 examined patients and the bile ducts in 77. The new contrast medium was also satisfactory in postcholecystectomy cases.

In view of the good results, a combination of the gallbladder examination with a gastrointestinal series was undertaken. At first, the examination was combined with 24 hour barium meal studies to rule out adhesions of the gallbladder to the colon. With the aid of erect and oblique views it was possible to eliminate a disturbing superimposition of the colon in all but 3 per cent of 298 consecutive cases.

Another method consisted of mixing granules of SH 771 with the barium meal. In order to avoid confusing images, the authors first performed a mucosal study of the stomach with barium alone and administered the mixture immediately afterward. In 13 out of 20 normal cases, the gallbladder filled in 2½ hours, in 3 cases it filled after 3½ hours and in the remaining 4 only after 24 hours. The bile ducts were visualized at 2½ hours in 75 per cent of the cases. Occasionally, there was a 24 hour delay of bile duct visualization.

Estimation of the relationship of the gallbladder to the stomach and duodenum was made possible. Accurate localization of the papilla Vateri with the combined method was of special interest. Out of a series of 150 cases, pathologic changes, not previously recognized, were detected in 52 patients. This number also includes calculous disorders in 15 per cent.—*Ernest Kraft, M.D.*

BUCHTALA, V. Die perorale Cholangio-Cholezystographie mit SH 771: Ergebnisse und

Nebenerscheinungen bei 766 Untersuchungen. (Peroral cholangio-cholecystography with SH 771: results and side effects in 766 examinations.) *Röntgen Blätter*, May, 1966, 19, 268-272. (Address: Nymphenburger Strasse 163, 8 München 19, Germany.)

A new contrast medium for oral cholecystography and cholangiography—SH 771 (Schering)—was tested in 566 cases. It was rapidly visualized in the gallbladder and bile ducts with optimal filling 3 hours following the ingestion. The findings were "positive" in 95 per cent of cases and "negative" in 5 per cent. Ducts only were visualized in 9 per cent. The cystic duct was filled in 85 per cent, the common duct in 77 per cent, and the hepatic duct in 59 per cent.

In a second series of 200 cases, the findings were similar except for the fact that calculi were detected in 20.5 per cent. Duct stones were observed in 8.5 per cent of the cases. The results in the 766 cases were far superior to those obtained with other contrast media.

In the second series of 200 cases, the patients were systematically questioned for possible side effects. Thus, a new type of reaction, an "internal chill," was reported in 80 per cent of the cases, while nausea and vomiting only appeared in 3.5 per cent. The sensation of a chill was predominant in the abdomen, but occasionally also in the long bones and the skin. Transient anesthesia of the head, lips, and forehead was encountered in 5 per cent of the cases. The side effects appeared most frequently when the contrast medium was ingested in the early morning hours. They could be alleviated by a different mode of administration or by a lower dose. Despite the somewhat disturbing reactions, the new drug proved superior and aided in rendering intravenous cholangiography almost completely avoidable.—*Ernest Kraft, M.D.*

## RADIATION THERAPY

JEFFERSON, A. Symposium on pituitary tumours: I. Clinical management and the results of treatment of the chromophobe pituitary adenoma. *Clin. Radiol.*, April, 1966, 17, 141-148. (From: Department of Neurological Surgery, The Royal Infirmary, Sheffield, England.)

The chromophobe pituitary adenoma is usually one of the most satisfying lesions that the neurosurgeon is called upon to treat. Good results should invariably follow early diagnosis and, with adequate treatment, life-long cures can be anticipated. Delayed diagnosis and late referral to the neurosurgeon are usually associated with extensive optic atrophy. As a result little if any improvement in vision follows treatment. Whenever optic atrophy has not destroyed the chance of improvement, restoration of visual function has been, for the majority of cases, both dramatic and speedy.



The operation is performed by cutting a window in the anterior wall of the lesion and by evacuating its contents. The surgeon aims at packing the capsule down into the floor of the pituitary fossa. The operation results in removing the pressure from the optic nerves and chiasm. The purely intrasellar lesions and those with a very large suprasellar lesion are recommended for radiation treatment only. Adenomata with a moderate sized suprasellar extension are treated by operation and subsequent irradiation. Among 51 patients there have been 4 deaths.

Every patient routinely receives a small dose of cortisone (12 mg. twice daily) and of levo-thyroxine sodium (0.1 mg. twice daily) for several years following treatment. Almost all the patients resumed work within 3 months of operation.

To date among the patients treated, with 60 per cent followed for 3 years, there has been no clinical or radiologic evidence of recurrence. If the recurrence rate in this series is eventually confirmed to be unusually low, perhaps this will be shown to have resulted either from the regular use of hormones or from excellent supervoltage roentgen irradiation.—*Samuel G. Henderson, M.D.*

LEWTAS, N. A. Symposium on pituitary tumours: II. Radiology in diagnosis and management. *Clin. Radiol.*, April, 1966, 17, 149-153. (From: Department of Radiology, United Sheffield Hospitals, Sheffield, England.)

While the most common pituitary tumor, the chromophobe adenoma, is traditionally described as producing a "ballooned sella" on lateral skull roentgenograms, this is actually the least common appearance. In nearly half of the patients with chromophobe adenoma there is found to be a double floor of the pituitary fossa due to asymmetric expansion by the intrasellar tumor. The extent of any suprasellar extension can be determined only by air encephalography. The anterior end of the third ventricle must always be clearly outlined with air. This is demonstrated by placing the head in the brow-up over-extended position. Tomography has proved especially helpful in improving the results. If satisfactory tomograms are not obtained at first, it may be possible to show the front of the third ventricle by delayed roentgenograms taken between 6 and 24 hours after air injection.

Effective treatment by either surgery or radiation therapy or both results in the majority of cases of pituitary adenoma, the patients living for many years, with rarely any recurrent tumor growth. Very large suprasellar extensions are unsuitable for direct surgery, and are treated by radiotherapy alone.

Since a lateral skull roentgenogram provides much information concerning the situation within the pituitary fossa, annual serial roentgenograms are desirable in follow-up. Alterations in the shape and thickness of the bony walls of the sella give reliable evidence of reduction of tumor size and of absence of recurrent tumor.

Serial lateral roentgenograms should be examined for (1) increase in thickness of the sellar floor and dorsum, (2) forward hinging of the dorsum, (3) forward and downward "crumpling" of the dorsum over the pituitary fossa, and (4) reduction of the vertical height of the dorsum.

By means of these signs the author has observed reduction of the tumor mass in 37 per cent of patients, the changes usually becoming evident between the 10th and 18th month after treatment. In 63 per cent of patients with no clinical evidence of tumor recurrence the roentgenographic appearances of the pituitary fossa remained unchanged.—*Samuel G. Henderson, M.D.*

EMMANUEL, I. G. Symposium on pituitary tumours: III. Historical aspects of radiotherapy, present treatment technique and results. *Clin. Radiol.*, April, 1966, 17, 154-160. (From: Sheffield National Centre for Radiotherapy, Sheffield, England.)

The name of Harvey Cushing stands out with unique distinction in the literature published on the subject of the pituitary in the first 30 years of this century. At the persuasion of Sosman many of Cushing's patients with chromophobe tumors received postoperative radiotherapy. Cushing was unconvinced of the real value of radiotherapy until Henderson (1939) reviewed Cushing's series and reported in great detail on 338 patients with pituitary tumors treated over the period 1913 to 1932. The finding that postoperative radiotherapy reduced significantly the incidence of recurrence did much to throw Cushing's weighty opinion in favor of postoperative irradiation.

Following the installation of a 2 mev. Van de Graaff generator at the Sheffield Center in 1948, the majority of pituitary tumors have been treated on this machine. A 5 field technique is used, a dose of 4,000 r in 4 weeks being delivered. With the exception of some of the eosinophil adenomas, all pituitary tumors are referred by the neurosurgeons after the necessary investigations and operation when indicated. An important contribution to the successful treatment of these tumors is the meticulous care with which the tracings of the tumor volumes are made by the neurosurgeons. The peak age incidence for chromophobe adenomas is in the 5th decade and for eosinophil adenomas in the 4th decade.

Sixteen patients were treated by radiation alone. Twelve of the 16 were free of recurrence at 4 years and more. Forty-six patients were referred for postoperative radiotherapy. Five died of postoperative complications, but 38 out of the remaining 41 were free of recurrence at 4 years and more.

In eosinophil adenomas radiotherapy will usually relieve the local pressure effects of the tumor. The systemic consequences of the endocrine disorder, falling mainly on the cardiovascular system and the carbohydrate metabolism, are relatively unaffected by treatment and lead to the early death of the pa-

tient. Acromegaly is arrested in a large proportion but improvement is rare. New interest in the management of this disease has been revived with pituitary ablation, either by surgery or by isotope implants or by "radio-cautery" using high energy particle beams.—*Samuel G. Henderson, M.D.*

WOLMAN, LIONEL. Symposium on pituitary tumours: IV. Unusual pathological features. *Clin. Radiol.*, April, 1966, 17, 161-168. (From Department of Neuropathology, The Royal Infirmary, Sheffield, England.)

In this paper material from 83 cases of anterior lobe tumors is reviewed and the range of histologic appearances illustrated. These tumors are discussed in 3 main groups according to the embryologic development of the main components of the gland: (1) pars glandularis or anterior lobe; (2) pars nervosa or posterior lobe; and (3) abnormal persistence of an intraglandular cleft between the pars glandularis and the pars nervosa, with development of a cystic tumor in or around the pituitary fossa.

The normal anterior lobe of the pituitary gland in the adult male is composed of 52 per cent chromophobe cells, 37 per cent eosinophils and 11 per cent basophils. Chromophobe adenomas are about four times more common than tumors arising from chromophil cells. The size of chromophobe tumors varies greatly from small intrasellar lesions to massive suprasellar extensions. They occur twice as frequently in the male as in the female. Pure eosinophil adenomas are rare, and only 1 such lesion was found in the 83 cases. Mixed tumors containing both eosinophilic and chromophobe cells are common. Basophil adenomas are considered to be secondary to endocrine disturbance arising in the adrenal. Primary tumors of the posterior lobe are rare. Of the tumors of the posterior lobe, a few gliomas have been described. Those tumors resembling in structure the infundibulum involve the pituitary stalk and adjacent floor of the third ventricle. The pathogenesis of craniopharyngiomas is attributed to small nests of squamous epithelium normally found in the pars tuberalis mainly above the diaphragma sellae. Such cell nests are not commonly found before the third decade.—*Samuel G. Henderson, M.D.*

ROBBINS, GUY F., LUCAS, JOHN C., JR., FRACCHIA, ALFRED A., FARROW, JOSEPH H., and CHU, FLORENCE C. H. An evaluation of postoperative prophylactic radiation therapy in breast cancer. *Surg., Gynec. & Obst.*, May, 1966, 122, 979-982. (From: Department of Surgery, Breast Service, and Department of Radiation Therapy, Memorial Hospital for Cancer and Allied Diseases and James Ewing Hospital, New York, N. Y.)

A quasi statistical comparison of treatment results

of breast cancer with and without prophylactic postoperative irradiation is given. The clinical material consists of 386 patients generally treated with radical mastectomy without radiotherapy during 1950 and 1951 and 349 patients usually treated with radical mastectomy plus postoperative irradiation during 1954 and 1955. There were exceptions to the treatment modalities in each group.

Since there were some patients treated with and without postoperative irradiation in both groups, the results were compared by two methods. The first was to calculate the results in groups who did or did not not receive prophylactic irradiation, regardless of their years of treatment. The second was to analyze the results in all patients of each 2 year group, regardless of whether they did or did not receive irradiation.

When radiotherapy was given, 3,500 rads mid-plane dose was delivered in 3-4 weeks using orthovoltage. Both the supraclavicular and internal mammary lymph nodes were usually treated. In general, the chest wall was not irradiated.

Of the 291 patients who received postoperative radiotherapy, 39 per cent were free of disease at 5 years. Among the 444 patients who did not receive radiotherapy, 58 per cent were without evidence of disease at 5 years. The authors indicate that there was selection favoring the nonirradiated group. There was no significant difference between the two methods of treatment if the axillary lymph nodes were negative. If the lymph nodes were positive, 29 per cent of the irradiated ones and 38 per cent of the nonirradiated ones were free of disease at 5 years. There was no difference in the survival rates if the patients were grouped according to the maximum level of axillary lymph node involvement.

Fifty-four per cent of the group treated in 1950 and 1951, usually without postoperative irradiation, survived 5 years without disease as compared to 47 per cent of the second group who were usually treated with postoperative irradiation.

Radiotherapy was found to be beneficial in reducing metastasis to the supraclavicular fossae. There was a 13 per cent incidence of metastasis to the supraclavicular fossae in 224 patients with positive axillary lymph nodes who received irradiation. The incidence rate was 26 per cent in the nonirradiated group of 200. Most of the metastases occurred during the first 18 months after operation in the nonirradiated group. Thereafter the occurrence rates were similar.

This is an uncontrolled study that seems to indicate that prophylactic postoperative irradiation is only beneficial in reducing the incidence of supraclavicular metastasis.—*Errett E. Smith, M.D.*

BUSCHKE, F. Some reflections of the treatment and prognosis of Hodgkin's disease. *Radiol. clin. et biol.*, 1965, 34, 285-309. (Address: Prof. Franz Buschke, University of Cali-



fornia, School of Medicine, San Francisco, Calif.)

The present optimistic view regarding the radiotherapeutic management of Hodgkin's disease is emphatically restated by the author, and illustrated by 3 cured cases surviving 30 years, 29 years, and 22 years, respectively. The value of the classification scheme, based on that described by Peters for management and prognosis, is supported.

Local recurrences of disease are attributed to inadequate dosage and the dose-dependent relationship of recurrent disease is reviewed. A dose of 3,500-4,000 r in 4-5 weeks is advocated as adequate to consistently avoid recurrences.

The question of radiation volume for early stage (Stage I and II) disease is discussed, as well as the therapy directed to apparent disease only or to extended fields (Peters and Kaplan).

The author reviews personal experience and tentatively concludes that regional treatment with "generous margins" is adequate for true Stage I disease, especially where only the cervical lymph nodes are involved. For Stage II disease, treatment of certain adjacent uninvolved areas, as well as an *en bloc* approach is advocated—the latter because of recurrences in treatment "gap" areas.

Chemotherapy has no place in Stage I or II disease, but finds useful application in situations where spinal cord compression is present. Generally, mediastinal disease can be treated by radiation therapy alone; however, where marked mediastinal obstruction is present, initial chemotherapy may be useful because of its more rapid action.

Stage III disease is considered for 2 situations: (1) Stage I or II disease that becomes Stage III by progression, and (2) widespread disease with a short history ("Class II"). The latter situation is handled by chemotherapy with irradiation reserved for selected areas of disease. Radiotherapy is integrated with other forms of therapy in the former situation.

The view that Hodgkin's disease is curable, as stated by Kaplan, Easson, and Peters, is supported by the author's experience.—*Yosh Maruyama, M.D.*

CONRADY, JUTTA, ELKIN, MILTON, ROMNEY, SEYMOUR L., and SANFILIPPO, LOUIS J. Pelvic angiography and lymphangiography in the evaluation of the patient with carcinoma of the cervix. *Surg., Gynec. & Obst.*, May 1966, 122, 983-990. (From: Departments of Radiology and Gynecology and Obstetrics, Albert Einstein College of Medicine of Yeshiva University, and the Bronx Municipal Hospital Center, New York, N. Y.)

In the management of carcinoma of the cervix, accurate determination of the extent of the disease is often difficult and may be erroneous.

Pelvic angiography and lymphangiography were performed on a group of Stage I and IIA cases following radiotherapy but prior to extraperitoneal lymph node dissection. The results showed that 4 of 7 patients had false positive lymphangiograms for lymph node metastases. In these cases, the studies added little useful information relating to the clinical status of the patient.

Use of pelvic angiography and lymphangiography in recurrent carcinoma may assist in the management of these cases.

In 2 cases reported, the pelvic angiogram delineated the size and extent of the pelvic tumor masses and enabled the authors to distinguish between an abscess and a recurrent tumor.

Twelve cases of more advanced carcinoma in clinical Stage IIB and IIIA were studied. In 3 of the 12 cases the angiograms and lymphangiograms provided additional data which led the authors to modify the planned management of these cases. One of the 3 cases was clearly overstaged and the other 2 had more extensive but clinically unsuspected disease.—*Edward M. Cockerill, M.D.*

LANG, ERICH K., NOURSE, MYRON H., WISHARD, WILLIAM NILES, JR., and MERTZ, JOHN H. O. The accuracy of preoperative staging of bladder tumors by arteriography: a 5-year study. *J. Urol.*, March, 1966, 95, 363-367. (From: Departments of Radiology and Urology, Methodist Hospital, Indianapolis, Ind.)

Preoperative staging of bladder carcinoma will determine the treatment modality to be used in the light of our present knowledge. Transurethral resection and electrosurgery are favored for papillomas with only minimal invasion. Segmental resection of the bladder is recommended for solitary well-localized lesions limited to the muscularis or penetrating into the fat capsule. Cobalt 60 teletherapy and supervoltage therapy are presently favored for multiple infiltrating lesions of Stage C or higher. Total cystectomy with radical lymph node dissection and by-pass procedures are now reserved for treatment of a few isolated instances of solitary but deeply infiltrating lesions.

Many methods have been used to evaluate the preoperative staging of the neoplasm. Bimanual examination under anesthesia, fractional biopsy, cystoscopy, intravesical and perivesical gas insufflation combined with perivesical venography and intravenous urography have all been accepted methods for study but each has inherent limitations in the accurate preoperative staging of vesical tumors.

The arteriographic demonstration of bladder tumors by the authors' method offers a reliable method for accurate and definitive assessment of the depth of infiltration of a bladder tumor and allows

certain conclusions regarding histologic grade of this tumor. One hundred and sixty-six patients with pathologic proof of bladder disease were studied by arteriography with an over-all accuracy in the diagnosis and staging of bladder tumors of about 90 per cent. In the Stage C tumor group, the accuracy was 95 per cent of 62 patients. The method was also extremely useful in followup examinations of Stage C bladder carcinomas treated by cobalt 60 teletherapy, and the over-all accuracy in this group was 94 per cent. The most difficult differential diagnosis was between carcinoma of the bladder Stage 0 and non-specific hyperemic cystitis.

The authors repeatedly emphasize the need for experience and familiarity with the method in order to establish a diagnostic accuracy of such high levels. The details of the arteriographic technique and positioning are included in this article.—*George W. Chamberlin, M.D.*

RUDD, KENNETH D., GREEN, ALLAN E., JR., MORROW, ROBERT M., and HANCOCK, JAMES A., JR. Radium source appliance for treatment of nasopharyngeal cancer. *J. Am. Dent. A.*, April, 1966, 72, 862-866. (From: Wilford Hall USAF Hospital, Aerospace Medical Division (AFSC), Lackland Air Force Base, Texas.)

The authors report a patient with a carcinoma of the nasopharynx for whom a radium source appliance was constructed.

The patient had a recurrence of her carcinoma of the nasopharynx 16 months after receiving 5,500 rads tumor dose with Co<sup>60</sup> teletherapy. It was decided to deliver 4,000 rads to the left nasopharyngeal fossa by the use of radium needles. A contour fitting acrylic resin appliance was constructed so that the needles would be positioned accurately. Similar appliances had been constructed in the past in which a cold-curing resin was adapted to the area of malignancy directly, and then removed for placement of the needles, in the operating room. This had the disadvantage of exposing the nasopharynx to the exothermic reaction of cold-curing acrylic resin, and inaccuracies resulting from construction of the appliance in the operating room.

The authors used a non-irritating polysulfide impression material to reproduce the area and then the appliance was constructed of heat-curing acrylic resin. The appliance fitted well and was well tolerated by the patient.

Although this procedure requires giving the patient an additional anesthetic, and requires more laboratory time than the one-stage procedure using cold-curing resin, it offers the advantage of precise adaptation, accurate placement of radium needles, and more comfort for the patient.—*Bernard Neff, M.D.*

BABINI, L., and SASSI, P. Perforazioni e fistole in corso di radioterapia dei tumori maligni dell'esofago. (Perforations and fistulae in the course of radiotherapy of malignant tumors of the esophagus.) *Radiobiologia, radioterapia e fisica med.*, Jan.-Feb., 1966, 21, 55-71. (From: Istituto di Radiologia dell'Università di Bologna, Bologna, Italy.)

Cancer of the esophagus in its progression, besides encroaching on the lumen, grows longitudinally and subsequently peripherally. It breaks through the muscle barrier and invades the mediastinum and its contents, frequently involving the tracheo-bronchial tree and producing perforations and fistulae.

The scope of the authors was to study in what way radiant therapy contributes to these complications.

The esophagus, in accordance with accepted radiologic convention, was divided in 3 portions:

(1) Upper third, which includes the cervical esophagus and the tracheal thoracic tract up to the aortic impression (32 cases).

(2) Middle third, which comprises the aortic narrowing, the tracheal tract below it and the infra-bronchial segment (71 cases).

(3) Lower third, which includes the pericardiac and abdominal portions (44 cases).

Complications generally occur a short time following the beginning of radiation therapy. In the majority of cases they take place during, or soon after the course of therapy. In 157 cases studied by the authors, only 3 presented esophago-tracheal or esophago-mediastinal fistulae some months after therapy.

Survival following the formation of fistulae is brief and coincides with the appearance of the complication.

The incidence of the complication is not dependent on the type of radiation used; *i. e.* 14.8 per cent for orthovoltage, 14.5 per cent for supervoltage therapy. There is no evidence that the dosage is a determining factor. It may occur with relatively low dosage.

It is logical to assume that radiotherapy rapidly destroying neoplastic tissue may accelerate perforation and formation of fistulae. The authors, however, conclude that the complication is not a direct consequence of radiotherapy.—*Anthony A. Blasi, M.D.*

ZUPPINGER, A. The importance of biology in radiotherapy. *Radiol. clin. et biol.*, 1965, 34, 310-322. (Address: Prof. Dr. A. Zuppinger, Zentralröntgeninstitut der Universität, Inselspital, CH-3000, Bern, Switzerland.)

The author defends the thesis that radiobiology is of importance to radiotherapy, and is essential to change radiotherapeutics from an empirical field to one based on scientific principles.

He reviews his work on calcium and phosphorus



metabolism of irradiated rat bone, measured by  $\text{Ca}^{45}$  and  $\text{P}^{32}$  uptake.

The results are considered in an uptake-removal model for bone mineral metabolism.—*Yosh Maruyama, M.D.*

MULLINS, FITZHUGH, MINTON, JOHN P., HOYE, ROBERT C., DEARMAN, JAMES R., and MCKNIGHT, WILLIAM B. The effect of high energy laser pulses on the primate liver. *Surg., Gynec. & Obst.*, April, 1966, 122, 727-732. (From: Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, and Applied Physics Branch, Redstone Arsenal, Huntsville, Ala.)

This investigation was suggested by the fact that McGuff, Minton, *et al.* were successfully able to destroy multiple tumor implants in experimental animals with high energy pulsed laser beams.

Multiple high energy laser impacts from a neodymium-doped glass laser with an energy range of 500 to 800 joules per impact were delivered to the surgically exposed livers of 12 healthy rhesus monkeys. All animals survived uneventfully and gross and histologic studies were done of the entire lesions at varying times (1 week to 3 months). Liver function tests were performed at frequent intervals up to 8 weeks, with only the serum lactic dehydrogenase values being markedly altered. These returned to normal in approximately 2 weeks.

The fact that the subhuman primate liver responded to the laser lesions by healing rapidly, without immediate or delayed hemorrhage and/or infection supports the belief that intensive studies are indicated to assess the value of laser radiation as a clinical entity in the treatment of metastatic hepatic neoplasms.—*Joseph Bissell, D.D.S.*

CADE, STANFORD. Iatrogenics. *Clin. Radiol.*, July, 1966, 17, 193-202. (From: The Westminster Hospital, London, England.)

The word "Iatrogenics" is defined by the author as "the study of diseases inflicted or created by doctors." Such diseases occur in the practice of all branches of medicine. Iatrogenic diseases following radiotherapy still occur. The early sequelae of exposure to radiation such as epilation and telangiectasis were followed with the advent of megavoltage therapy by atrophy of the subcutaneous tissue, delayed necrosis of the subcutaneous tissue, skin, muscle, and bone, and by visceral damage.

The most frequent site of subcutaneous fibrosis and necrosis is seen in the neck following repeated treatments, and in the pectoral and scapular regions and the axilla following radiotherapy for breast cancer. Loss of function is progressive, beginning about 12 to 18 months after treatment and increasing dur-

ing subsequent years. Most of the sequelae follow repeated treatments and tissue doses of over 5,000 r.

The deleterious effects on bone can be divided into 4 groups: (1) arrest of growth, (2) massive necrosis, (3) osteoporosis and fracture, and (4) post-radiation sarcoma.

The dangerous effects of radiation on the viscera have been known since early in the use of roentgen rays and radium, but it is only in the past 20 years that serious studies of the severe iatrogenic effects of radiation on the viscera have been published. Renal damage may follow abdominal irradiation for many conditions, particularly malignant tumors of the testis and abdominal lymphosarcoma. Radiation nephritis can develop within 6 to 12 months after radiotherapy, and present as an acute form, or years later as a chronic nephritis, followed by benign or malignant hypertension. Acute anuria may develop due to mechanical blockage of the ureters, renal pelvis and tubules by uric acid crystals following radiotherapy for abdominal lymphosarcoma.

In the lungs the bulk of the cases of radiation damage follow treatment for breast cancer. The use of low voltage, opposing fields, high dose and short time is conducive to radiation pneumonitis, fixation of the shoulder in some, fractured ribs in others, and always telangiectasis over the scapula and the pectoral area.

Myelopathy affecting the spinal cord and brain may follow radiation therapy. The safety limits of dose to the spinal cord should not exceed 3,300 rads for 30 cm. fields and 4,300 rads in smaller fields over a period of 42 days. Lesions to the brain following radiotherapy of brain tumors and neoplasms involving the base of the skull arising in the middle ear or nasopharynx may occur. A dose not exceeding 4,000 r is considered a reasonable risk.

The value of radiotherapy in the treatment of cancer of the cervix is unquestioned. The severe effects of radiation on the rectum and sigmoid include necrosis, perforation with fistula formation and stenosis requiring colostomy. Megavoltage has not eliminated these risks. Necrosis of the irradiated colon with abscess formation can occur within 6 months following megavoltage radiotherapy; stenosis may occur several years later. In the small bowel, bleeding, perforation, obstruction and fistulae have all been observed as sequelae to abdominal irradiation. The bowel damage, as in other structures, is due to vascular changes.

Cancer of the thyroid in children is now recognized with increasing frequency. In a series of 562 children with thyroid cancer, 80 per cent were found to have received irradiation in infancy or childhood for various conditions such as actual or presumed enlarged thymus, hypertrophied tonsils and adenoids, acne and nevi.

The incidence of hypothyroidism following  $\text{I}^{131}$  has been variously reported. It has varied from 11 per cent to 44 per cent according to the length of the

period of observation, the number of patients in the series and the dose of  $I^{131}$  given.

Leukemia may develop in children of mothers subjected to diagnostic radiologic procedures during pregnancy. The death rate from leukemia in patients given radiation treatment for ankylosing spondylitis is reported as 10 times that of the normal population. The risk is related to the dose of radiation, the age of the patient, and repetition of treatments.

Many types of human cancer have been produced by exposure to radiation. Consequently, benign lesions treatable by other means should not be treated by radiation. Radiotherapy in children should be limited to the treatment of cancer, *with no exceptions.*—*Samuel G. Henderson, M.D.*

RIGBY, CAROLYN C. The pathology of rhabdomyosarcoma in children. *Proc. Roy. Soc. Med.*, May, 1966, 59, 411-412. (From: Department of Morbid Anatomy, The Hospital for Sick Children, Great Ormond Street, London, England.)

The author reviews all cases of embryonic rhabdomyosarcoma diagnosed histologically at The Hospital for Sick Children over a 40 year period. This tumor accounted for 3.6 per cent of all tumors diagnosed during that period.

Several interesting conclusions are arrived at, among which are the following: commonest site of this tumor was the bladder; it occurred either in the urogenital organs or in the head; before it becomes invasive, early urinary obstruction may lead to the diagnosis; a characteristic macroscopic feature of rhabdomyosarcoma is its polyp-like formation; microscopically the tumor tissue consists of mesenchymal cells and more differentiated elements—fibrous tissue, striated and nonstriated muscle; metastasis to the lungs occurred more frequently than did metastasis to the bones.—*Lionel W. Young, M.D.*

#### RADIOISOTOPES

LOKEN, MERLE K., WIGDAHL, LUTHER O., GILSON, J. MICHAEL, and STAAB, EDWARD V. Mercury-197 and mercury-203 chlormerodrin for evaluation of brain lesions using a rectilinear scanner and scintillation camera. *J. Nuclear Med.*, March, 1966, 7, 209-218. (From: University of Minnesota and Veterans Administration Hospitals, Minneapolis, Minn.)

The authors summarize their experience in cephaloscanning of 432 patients conventionally scanned with  $Hg^{197}$  tagged chlormerodrin. Forty-two selected patients were studied with both  $Hg^{197}$  and  $Hg^{203}$  tagged chlormerodrin as the radioactive agent. Eight positive scans in this group were obtained by both isotopes. It is concluded that there is no significant difference in the value of these agents for demon-

strating brain lesions, but  $Hg^{197}$  tagged chlormerodrin is preferred because of smaller radiation to the kidneys.

There were 107 patients studied with rectilinear scanner and scintillation camera using  $Hg^{197}$  tagged chlormerodrin. Twenty-two positive camera studies were recorded. On 2 occasions the cameras recorded positive scans which appeared inadequate in rectilinear scanning because of poor patient cooperation. Twenty-nine positive rectilinear scans were obtained and 3 scans were positive that were not positive in the camera group.

The advantages and disadvantages of both the rectilinear scanner and the scintillation camera are discussed and it was felt that each gave information of essentially equal accuracy.—*Abbas M. Rejali, M.D.*

SCHMUKLER, MORTON, and WORKMAN, JOSEPH B. The reliability of scintillation scanning for detection of intracranial lesions. *J. Nuclear Med.*, April, 1966, 7, 252-258. (From: Department of Medicine, Radioisotope Laboratory, University of Maryland School of Medicine, Baltimore, Md.)

This report concerns an experience with 522 scintigrams done for the detection of intracranial lesions. The usual tracer was  $Hg^{203}$  labeled chlormerodrin, but a smaller recent experience has been accumulated with  $Hg^{197}$  chlormerodrin. Among those patients scanned for the detection of non-neoplastic disease were 115 thought to have vascular lesions. In only 8 per cent (9/115) were positive records obtained, a somewhat lower figure than one can get from other laboratories. Accuracy of scanning localization of primary tumors was 84 per cent. Seventeen of 22 astrocytomas of the various grades were detected, and all of 11 meningiomas. Twenty-one of 43 presumed metastatic lesions were found, including 13 of 16 proven ones.

The authors' data support the contention that radioisotope scanning is an excellent radiographic test for the detection of intracranial lesions, especially neoplasms.—*Frederick J. Bonte, M.D.*

GREENBERG, WAYNE V. Thyroidal  $I^{131}$  turnover in hypothyroidism: correlation with thyrotropin responsiveness. *J. Clin. Endocrinol. & Metabol.*, May, 1966, 26, 559-565. (From: Clinical Investigation Unit, and Metabolism Division, Department of Medicine, Medical College of Georgia, Augusta, Ga.)

Primary or thyroidal myxedema can be distinguished from secondary or pituitary myxedema by the response of the thyroid gland to exogenous thyroid stimulating hormone (TSH) or by the direct measurement of the circulating TSH. The present study was undertaken to test the hypothesis that the thyroidal  $I^{131}$  release would be a good index to distinguish



between the two types of myxedema. The thyroidal  $I^{131}$  release is influenced by endogenous TSH and is easily measureable.

Twenty-four hypothyroid cases were studied. Tracer doses of 100–200  $\mu\text{C}$  of  $I^{131}$  were given and counts were made for 7 days. The thyrotropin stimulation study was done after completion of the counting on the seventh day. Three injections of thyrotropin, each containing 10 U, were given and these were followed by a repeat 24-hour  $I^{131}$  study.

The protein bound iodine (PBI) study was higher in pituitary dysfunction than in primary hypothyroidism due, most likely, to the intrinsic thyroid pathology producing a greater hypofunction than the relative lack of thyrotropin. The  $I^{131}$  uptakes were higher in pituitary hypofunction as the thyrotropin loss was probably incomplete and the decrease in function was not as complete as that caused by a destructive process within the thyroid gland. The response to the TSH or thyrotropin separated the myxedematous cases in to two distinct groups.

The rate of  $I^{131}$  release, as demonstrated by the daily counts, was decidedly different between the two groups, and the difference was significant as early as 1 day following the standard 24-hour uptake study. There was further accumulation of  $I^{131}$  during the second 24-hour period following the tracer in the hypopituitary cases as compared with a marked loss in the primary myxedema cases during the same period.

The thyroidal  $I^{131}$  turnover was found to be a useful test in differentiating the two types of myxedema. A rapid release of thyroidal  $I^{131}$  supports the diagnosis of primary thyroidal disease, whereas a slow or absent release of  $I^{131}$  indicates a pituitary hypofunction. The accompanying TSH stimulation tests agreed with the thyroidal  $I^{131}$  turnover results.—Charles W. Cooley, M.D.

MORRIS, J. G., DOUST, B. D., SMITANANDA, N., WAGNER, P., and McRAE, J. Lung scanning—technique and some diagnostic uses. *Australasian Radiol.*, Feb., 1966, 10, 17–38. (From: Department of Medicine, University of Sydney, Sydney, Australia.)

Lung scanning was developed as an aid in diagnosing pulmonary embolism. However, abnormal scans were seen in a number of disease states. The authors studied 36 cases with the following clinical diagnoses: Normal (4), pulmonary embolism (15), primary lung carcinoma (9), secondary lung carcinoma (8), pulmonary artery agenesis (1), pulmonary hydatid disease or loculated effusion (1), tuberculoma (1), bronchopneumonia (1), and chronic obstructive airway disease (1). Liver-lung scans were done in 2 cases.

A scanner with a 3×2 inch crystal and a 19-hole collimator was used. The scans were done with the patient being either prone or supine, and at times in

both positions. The lung scans were best with the patient being prone, as a greater volume of lung was closer to the scanner. The supine lung scan was indicated when the patient was too ill to be placed prone, or when the pathology was located anteriorly. The scans were done immediately after the intravenous injection of 100–300  $\mu\text{C}$  of  $I^{131}$  macroaggregate human serum albumin. The scanning speed was 40–50 cm. per minute. The average scan lasted 30–40 minutes. The thyroid gland was blocked, when possible, by the administration of Lugol's solution.

Chest roentgenograms are essential prior to scanning. The interpretation of the scans is most satisfactory when combined with the clinical data and chest roentgenograms. Cardiac enlargement does make interpretation difficult, and especially when pathology is present in the left lung field.

The combined liver-lung scan was done by administering 100  $\mu\text{C}$  of colloidal  $\text{Au}^{198}$  intravenously 3 days prior to the scan. The liver-lung scan is of value in investigating right sided diaphragmatic abnormalities. Subphrenic lesions may distort the upper edge of the liver and the base of the lung.

There were 7 positive scans in the 15 clinically suspected cases of pulmonary embolism. Serial lung scans can be used to follow changes in pulmonary circulation during the natural and treated course of pulmonary embolism. Lung scanning may become a major indication for the selection of patients who require anticoagulant therapy or vena cava plication.

There were no constant findings in the lung scans of patients with primary and secondary lung carcinoma. Some scans were normal and some showed the same or greater changes when compared with the chest roentgenograms.

Normal and abnormal scans are presented to illustrate various disease states and points of technique.

Lung scanning with  $I^{131}$  macroaggregated human serum albumin was found to be a safe procedure. The scan abnormalities are produced by diseases or abnormalities causing obstruction of pulmonary arteries or an alteration of the pulmonary arterial blood flow.—Charles W. Cooley, M.D.

TAPLIN, G. V., POE, N. D., and GREENBERG, A. Lung scanning following radioaerosol inhalation. *J. Nuclear Med.*, Feb., 1966, 7, 77–87. (From: Laboratory of Nuclear Medicine and Radiation Biology, School of Medicine, University of California at Los Angeles; and Department of Radiology, Los Angeles County Olive View Hospital, Olive View, Calif.)

The authors describe the development of a safe radioaerosol inhalation procedure and lung scanning technique using standard respirator-nebulizer equipment and minute quantities of radioisotopes. The

various available radioaerosols are described along with their pulmonary radiation exposures and mode of absorption. Economically colloidal  $\text{Au}^{198}$  is the most suitable agent. It is removed rapidly from the lung mainly by ciliary action and is not absorbed from either the lung or the intestine. However, because its energy is so similar to  $\text{I}^{131}$  it cannot be used on the same day with  $\text{I}^{131}$ HSA macroaggregates for combined studies of bronchial patency and regional blood flow by lung scanning. For this purpose aerosols of  $\text{Hg}^{197}$  chlormerodrin or  $\text{Tc}^{99m}$  labelled albumin are preferable.

Correlating the experimental work in dogs with the observations made on 75 patients with various lung disorders, the authors came to the following conclusions:

1. Under normal conditions the entire lung field is visualized indicating the patency of the lower bronchial tree.
2. Complete obstruction of a major bronchus produces absence of radioactivity in the region normally ventilated by this bronchus.
3. The amount of radioaerosol deposited in any given portion of the lung is not always proportional to the air flow to that region. Factors, other than diffusion, like particle size, sedimentation impaction, concentration of the aerosol, and rate of air movement play a very important role. When inhalation is performed with the subject in lateral position, greater amounts are deposited in the superior lung where ventilation and perfusion are reduced.
4. With partial obstruction increased deposition of radioaerosol may occur at the obstruction site due to impaction, and the region beyond obstruction usually shows reduced level of radioactivity.
5. Pulmonary embolism is not detected by inhalation technique but later when collapse of a segment of lobe occurs, it will show reduced activity.

The radioaerosol inhalation procedure is a useful adjunct to bronchography and can be safely employed where bronchography is contraindicated.—*Surrendra K. Bansal, M.D.*

SHEHADI, WILLIAM H. Practical applications of liver scanning. *Radiology*, April, 1966, 86, 726-733. (Address: Department of Radiology, United Hospital, Port Chester, N. Y.)

A series of 134 patients examined by liver scanning is reviewed and the pertinent findings presented. Rose bengal  $\text{I}^{131}$  and colloidal  $\text{Au}^{198}$  were the isotopes used for the study, with the choice of isotope and the procedure employed depending upon the history and clinical findings.

$\text{Au}^{198}$  was used when the following determinations were to be made: (1) size, shape, contour, and position of the liver; (2) the presence of a space-occupy-

ing lesion, be it neoplastic, inflammatory, or otherwise; (3) the relation of an abdominal mass to the liver; and (4) the status of the liver, as part of a routine preoperative survey, in known cases of neoplastic disease.

Rose bengal  $\text{I}^{131}$  was used for study of liver function and the differential diagnosis of jaundice. Also, liver scanning following the simultaneous injection of both these isotopes was performed in patients with known malignant disease in whom jaundice had developed.

$\text{Au}^{198}$  was employed in 90 patients and Rose bengal  $\text{I}^{131}$  in 44—a ratio of 2:1. Of those receiving Rose bengal  $\text{I}^{131}$ , there was normal flow into the intestines in 11, and obstructed flow in 33. Two of these had partial obstruction to the flow of bile; the remaining 31 were totally obstructed. Among the 90 patients receiving  $\text{Au}^{198}$ , there were 47 normal and 43 abnormal scans.

The results of the study together with the numerical break-down of abnormalities are presented in tabular form.—*Howard R. Stewart, M.D.*

CAVALIERI, RALPH R., SCOTT, KENNETH G., and SAIRENJI, EIKO. Selenite ( $74\text{ Se}$ ) as a tumor-localizing agent in man. *J. Nuclear Med.*, March, 1966, 7, 197-208. (From: Radioisotope Service, Veterans Administration Hospital, San Francisco, and Radioactivity Research Center, University of California, San Francisco Medical Center, San Francisco, Calif.)

Observations made by one of the authors (K.G.S.) on rats bearing sarcoma suggested that  $\text{Se}^{75}$  selenite ( $\text{SeO}_3^{2-}$ ) concentrates selectively in the tumor.

Using  $\text{Se}^{75}$  labeled selenite in human, intracranial as well as intrathoracic and intra-abdominal neoplasms were successfully localized by scintillation scanning.

The doses of  $\text{Se}^{75}$  ranged from 1.5 to 4  $\mu\text{C}/\text{kg}$ . given intravenously and the patients were scanned at intervals of 4 to 96 hours.

The assays of specimens of tumors obtained at operation or autopsy revealed concentrations of  $\text{Se}^{75}$  ranging from 0.8 to 4.6 times the level of plasma.

Distribution studies showed a relatively high concentration of  $\text{Se}^{75}$  in the liver and kidneys but a low concentration in the skeletal muscle.

The total body radiation from a dose of 4  $\mu\text{C}/\text{kg}$ . body weight calculated on the basis of average effective  $T_{1/2}$  of 43 days (for 75 per cent of the administered dose) was 1.14 rads.

It has been stated that  $\text{Se}^{75}$  selenite may offer specific tumor localizing properties not found in other currently used agents. This agent appears to be useful in differentiating cerebrovascular from the other type of intracranial lesions.—*Abbas M. Rejali, M.D.*



DeNARDO, GERALD L., and VOLPE, JOSEPH A. Detection of bone lesions with the strontium-85 scintiscan. *J. Nuclear Med.*, March, 1966, 7, 219-236. (From: Radioisotope Service, Veterans Administration Hospital, Stanford School of Medicine, Palo Alto, Calif.)

The authors reviewed their experience with 164  $\text{Sr}^{85}$  bone scintiscans. These scans were performed in 50 patients on 59 occasions.

Thirty-two of 50 patients had an abnormal bone scan. This included 17 with metastasis, 9 with osteomyelitis, 2 with lymphoma, 2 with primary bone neoplasm, 1 with Gaucher's disease and 1 with Paget's disease. Fourteen of these patients had positive scans and negative roentgenograms at the time of the scanning.

The technique appeared to be valuable especially in the study of localized bone disease such as early bone metastasis and osteomyelitis and in differentiating bone islands from osteoblastic metastasis.

Also, preliminary remarks are made on the use of the whole body scanner, consisting of 2 above and 2 under the table scintillation detectors, as a rapid screening method for evaluation of skeletal disease and to obtain a count rate profile of the areas of increased  $\text{Sr}^{85}$  concentration.—*Abbas M. Rajali, M.D.*

GOTTSCHALK, ALEXANDER, HARPER, PAUL V., JIMINEZ, FELICIANO F., and PETASNICK, JERRY P. Quantification of the respiratory motion artifact in radioisotope scanning with the rectilinear focused collimator scanner and the gamma scintillation camera. *J. Nuclear Med.*, April, 1966, 7, 243-251. (From: Department of Radiology, and the Argonne Cancer Research Hospital, University of Chicago, Chicago, Ill.)

Unsharpness of the radiographic image as the result of respiratory motion is a feature of a number of important radioisotope tests, including the liver scan. Using the Siemens "sign-wave phantom" which features alternate, radial, active and inactive rays, the authors reproduced respiratory motion with a moving platform system. The effects of motion of this system were tested on the radiographic images produced by a scintillation camera, a commercial rectilinear scanner, and a custom built high-speed rectilinear scanning system. In all cases,  $\text{Tc}^{99m}$  was used as the emitter. The effects of respiratory motion are well shown in reproduced scans made with the various instrument systems, showing the phantom both at rest and moving.

Improvement in radiographic quality of the scintillation camera image with a technique of suspended respiration is illustrated.—*Frederick J. Bonte, M.D.*

DAVIS, THOMAS P., and MARTONE, RONALD J. The hybrid radioisotope scanner. *J. Nuclear*

*Med.*, Feb., 1966, 7, 114-127. (From: Department of Radiation Biology and Biophysics, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.)

A radioisotope mapping system, intermediate in speed and complexity between a standard mechanical rectilinear scanner and a stationary camera-type, was developed in the authors' laboratory. In this new device, the distribution of activity in the transverse direction across the subject is translated into a corresponding distribution of detected events in a long rod-shaped detector. This latter distribution is then sensed electronically. The complete area map is generated by mechanically moving the subject longitudinally, in a direction perpendicular to the long axis of the detector. Because of this combination of an electronically sensed transverse scan with a mechanical longitudinal scan, the instrument has been called the hybrid scanner.

In this paper, the authors discuss the operating principle and design of the hybrid scanner. The principle is based on the observation that when a scintillation event occurs in a long rod of fluor, the logarithm of the ratio of the fluorescent radiation fluxes issuing from opposite ends of the rod, to a close approximation, is directly proportional to the position of the scintillation event in the direction parallel to the long axis of the rod.

Several clinical scans are presented, including a brain scan made in 90 seconds under unusually favorable conditions of high activity and high target-to-nontarget specific activity ratio. The scanner has proved to be extraordinarily simple to align and operate, and has produced radioisotope maps of satisfactory quality in about one-half to one-fifth of the time required for a conventional rectilinear scan.—*Surrendra K. Bansal, M.D.*

HORWITZ, N. H., LOFSTROM, J. E., and FORSAITH, A. L. A comparison of clinical results obtained with a spark-imaging camera and a conventional scintillation scanner. *Radiology*, May, 1966, 86, 830-834. (Address: 3601 West 13 Mile Road, Royal Oak, Mich.)

The authors describe the use of a spark-imaging camera, the spintharicon, in over 75 patients in whom the thyroid gland was being evaluated. Spark images obtained consecutively together with conventional scintiscans 24 hours after the administration of 100  $\mu\text{C}$   $\text{I}^{125}$  permitted the evaluation of the clinical usefulness of this instrument. The results from this series of patients indicated that the spark-imaging camera consistently produced distribution patterns of higher resolution and greater detail in about one-half the time required for a scintiscan.

It was found that the gradation of tones available in the spark image permitted visualization of subtle features in the distribution pattern. The composition

of the spark image also more correctly displayed the true nature of the glandular structure and function than did the interrupted pattern of the line scan. Artifacts inherent in the scintiscan image, a consequence of the line-by-line process by which the image is constructed, are not evident with the use of the spintharicon since no scanning process is required.

The principle of the spark-imaging camera is briefly discussed and the manner in which the camera is used is presented.

The authors conclude that the simplicity, freedom from artifacts, and the low cost suggest that this may be an ideal instrument for applications where low-energy gamma emitters are employed.—*Donald N. Dysart, M.D.*

DUGGAN, H. E., and WEIJER, D. L. Polycythemia rubra vera and radioactive phosphorus—90 patients. *J. Canad. A. Radiologists*, March, 1966, 17, 4-9. (Address: Dr. Duggan, Director, Department of Radiology, Foothills Hospital, Calgary, Alberta, Canada.)

A series of 90 patients with polycythemia vera treated with radioactive phosphorus is reported. Ninety per cent of the patients received 3 doses or less of  $P^{32}$ , with average dose per treatment of about 5 mc. It is stated that the dosage need not be increased if the phosphorus is given orally (*J. Canad. A. Radiologists*, 1962, 13, 81).<sup>\*</sup> At the end of 10 years, 70 of the 90 patients were still living. The most common cause of death was thrombotic disease, but 3 patients died of chronic myelogenous leukemia and myelofibrosis developed in 5 patients who are still living. No cases of acute leukemia occurred in the series. The authors feel that the late onset of myelofibrosis supports the theory that this represents a late stage of polycythemia.

It is contended that the therapeutic effect of  $P^{32}$  in polycythemia vera is due to fragmentation of the DNA molecule after incorporated  $P^{32}$  transmutes to  $S^{32}$ , rather than to the accompanying beta radiation. As evidence for this they cite the fact that peripheral blood cultures irradiated with  $P^{32}$  show a higher incidence of nuclear damage than do cultures treated with external irradiation.—*E. A. Franken, Jr., M.D.*

### CHEMOTHERAPY

FRECKMAN, HERMAN A., MENDEZ, FERNANDO L., JR., MAURER, ELMER R., and FRY, HARRY L. Chemotherapy for lung cancer in intra-aortic infusion. *J.A.M.A.*, April 4, 1966, 196, 5-10. (From: Departments of Oncology and Surgery, Christ Hospital and Bethesda Hospital, Cincinnati, Ohio.)

<sup>\*</sup> Remission time was noted to be longer in those patients receiving phlebotomies prior to administration of phosphorus.

The authors describe a method of administering 5-fluorouracil and cyclophosphamide to tumor bearing areas in patients with bronchogenic carcinoma or metastatic lung cancer.

A polyethylene catheter is positioned in the aorta just beyond the origin of the bronchial arteries. Continuous infusions are then carried out for at least a period of 10 to 14 days.

There were only minimal complications attributed to this technique.

Both groups of patients exhibited similar survival times and durations of the objective response in bronchogenic carcinoma, while the objective response rate was 3 times higher in the cases of metastatic lung cancer.—*Henry Haven, M.D.*

### MISCELLANEOUS

MACKAY, JOHN S., and ROSS, RODERICK C. Hepatoma induced by thorium dioxide (thorotrast). *Canad. M. A. J.*, June 18, 1966, 94, 1298-1303. (From: School of Hygiene, University of Toronto, and Department of Pathology, St. Michael's Hospital, Toronto, Ontario, Canada.)

In spite of warnings of the oncogenic potential of thorium dioxide as early as 1932, widespread use of this agent in a variety of radiographic procedures continued until about 1956.

The authors describe a case in which the patient gave no history of thorium dioxide administration. Roentgen examination revealed multiple, irregular radiodensities in the spleen, liver, and mid-epigastrium, and an osteolytic lesion in the sacrum. Microscopic studies of liver specimens showed portal cirrhosis and necrosis. Hemorrhage, brown pigmentation, and fibrosis with much granular material were noted in the spleen. Autoradiography of the spleen revealed short linear tracks interpreted as alpha tracks, thus confirming the assumption that the roentgenographic densities and microscopic granular material was thorium dioxide.

Most of the isotope remains in the body for life and forms aggregates of various sizes. Although this radioactive agent yields alpha particles of short range, such radiation has a high specific ionization and the adjacent tissues receive large total amounts.

It has become apparent that thorium dioxide induced tumors develop after long latent periods, Dahlgren having reported an average latent period for liver tumors of 18 years, with a range of 3 to 34 years. It is probable that more patients with these neoplastic lesions will be seen during another decade or so.

It is, therefore, important to be aware of the condition and to be familiar with the roentgen and histologic appearances.—*Lois Cowan Collins, M.D.*



BIZZOZERO, O. JOSEPH, JR., JOHNSON, KENNETH G., and CIOCCO, ANTONIO. Radiation-related leukemia in Hiroshima and Nagasaki, 1946-1964: I. Distribution, incidence and appearance time. *New England J. Med.*, May 19, 1966, 274, 1095-1101. (From: Atomic Bomb Casualty Committee, Hijiya Park, Hiroshima, Japan.)

This report summarizes the data of the Atomic Bomb Casualty Committee regarding detection and incidence of leukemia in Hiroshima and Nagasaki between 1946 and 1964. It affirms the role of ionizing radiation in leukemogenesis.

During the 18 year interval, 1,089 cases of leukemia were recorded, of which 738 patients were born prior to the bombings in August 1945. The distance in meters from the bomb's hypocenter was used as an index of exposure.

In the more heavily irradiated group (exposed within 1,500 meters) the appearance time of acute leukemia in patients under 30, and of chronic leukemia in all age groups, was more rapid than in the less heavily exposed group. It is hypothesized that age is a co-variable to distance in the appearance time of acute leukemia.

Chronic granulocytic leukemia developed in heavily irradiated persons up to 29 years of age at the time of the bombings at a disproportionate rate as compared to the distally exposed and the pattern of spontaneous leukemia for this age group.—*Lois Cowan Collins, M.D.*

CONARD, R. A., RALL, R. E., and SUTOW, W. W. Thyroid nodules as a late sequela of radioactive fallout in a Marshall island population exposed in 1954. *New England J. Med.*, June 23, 1966, 274, 1391-1399. (From: Medical Research Center, Brookhaven National Laboratory, Upton, N.Y.)

Pathologic changes in the thyroid gland attributable to radioactive fallout were detected in a number of Marshallese people.

No thyroid pathology was noted in 200 individuals of a control group who were not exposed to the radiation.

In the most heavily exposed population (175 rads whole-body gamma radiation) thyroid abnormalities developed in over one-half of the children under the age of 10 years. Five children had benign

adenomatous goiters, the one adult patient had a mixed papillary and follicular carcinoma with localized metastasis.

The data indicate the caution needed with the clinical use of radiation, particularly in children.—*Lois Cowan Collins, M.D.*

WARD, GERALD M., JOHNSON, JAMES E., and WILSON, DANIEL W. Deposition of fallout cesium 137 on forage and transfer to milk. *Pub. Health Rep.*, July, 1966, 81, 639-645. (From: Department of Animal Science and Radiation Biology, Colorado State University, Fort Collins, Colo.)

Cesium 137 is one of the most important contaminants from fallout nuclear debris because of its long physical half-life and affinity for biologic systems. Body burdens of this radionuclide in man result principally from the food-chain sequence: air and precipitation to plants, plants to milk and meat, with dairy and beef cattle as the principal vectors between plants and man's diet. Mathematical models describing the transport of radioactive fallout to milk, emphasizing strontium 90, have been presented. The authors report their observations on the transport of cesium 137 in a controlled dairy ecosystem at Colorado State University. The study began in 1962 and was carried out on a herd of 112 cows and an irrigated farm in a geographic region that would be classified as a dry area.

Milk samples were collected from 12 cows on pasture and 100 cows fed on dry lot in 1962, 1963, and 1964. Concurrent samples of food intake and milk production from each cow, daily air samples, and samples of each rainfall were collected and analyzed for cesium 137 content. The deposition of cesium 137 in rainfall ranged from 300 to 7,000 picocuries per square meter and on alfalfa hay from 60 to 1,640 picocuries per square meter. Deposition coefficients ranged from 0.06 to 0.37. The maximum levels of cesium 137 in milk from cows fed on pasture were about 200 picocuries per liter in 1962 and 1963, while the maximum levels in milk from cows fed on dry lot were about 170 picocuries per liter. Levels of cesium 137 in milk were lowered during the winter months because third-cutting hay with low cesium 137 activity usually was fed to all the cows. Transfer coefficients, the percentage of ingested cesium 137 found per liter of milk, were higher for high-grain diets (0.58) than for predominantly hay diets (0.24).—*Luther W. Brady, M.D.*



# SUBJECT INDEX TO VOLUME 98

(ab) = abstract

(E) = editorial

- ABDOMINAL aortic aneurysms, catheter arteriography in evaluation of, 879
- masses, value of nephrotomography in differential diagnosis of, 935
- viscera, tomographic exploration of, by combined pneumoperitoneum and retroperitoneum (ab), 1019
- ACROMEGALY, therapy of, transnasal-transsphenoidal yttrium-90 pituitary implantation in (ab), 774
- ADENOMAS, pituitary, extrasellar extension of; clinical and neuroradiological considerations, 616
- ADRENAL angiography, selective (ab), 262
- ADULT PEDIATRIC CHEST UNIT, combination, 492
- AGENESIS of corpus callosum, angiographic findings in, 579
- AMERICAN ROENTGEN RAY SOCIETY, sixty-seventh annual meeting of (E), 980
- AMNIOTIC FLUID, opacification of, for localization of placenta (ab), 516
- pulmonary embolism, nonfatal, 434
- ANASTOMOSIS, pharyngeal-vertebral, ascending, 599
- ANEMIA, delay in diagnosis of carcinoma of right side of colon in patients treated for (ab), 259
- ANEURYSM as cause of stroke, 647
- of thoracic aorta, an aid in diagnosis of; catheter position, 836
- ANEURYSMS, abdominal aortic, catheter arteriography in evaluation of, 879
- multiple bilateral intracranial intracavernous and intradural carotid: compendium of classical findings—case report (ab), 1014
- renal artery, 132
- sinus of Valsalva (ab), 258
- ANGIOCARDIOGRAPHIC diagnosis of pericardial effusion, 874
- ANGIOCARDIOGRAPHY, osmotic effects of urografin 76 per cent and isopaque 60 per cent in, 922
- ANGIOGRAM, pulmonary, and pulmonary embolism, observations on, 859
- ANGIOGRAPHIC features of primary venous obstruction of upper extremity; Paget-von Schrötter syndrome, 388
- findings in agenesis of corpus callosum, 579
- findings in patient with tuberous sclerosis, 723
- ANGIOGRAPHY, adrenal, selective (ab), 262
- carotid, possibilities for incorrect diagnosis of brain abscess by means of (ab), 769
- cerebral, possibilities for error in (ab), 768
- of pheochromocytoma, 225
- of posterior cervicocranial circulation, 660
- of renal agenesis and dysgenesis, 137
- post, isotope brain scanning; positive or negative? 672
- renal, subtraction technique in (ab), 773
- subclavian and internal mammary, in evaluation of anterior mediastinal masses, 447
- ANGIO-RENOGRAPHIC studies in oestrone-pretreated adrenalectomized and hypophysectomized rats following administration of posterior pituitary extract (ab), 520
- ANGIOTENSIN, effects of, on renal vascularity in dogs, 927
- ANTERIOR MEDIASTINAL masses, subclavian and internal mammary angiography in evaluation of, 447
- ANUS, imperforate, association of lumbosacral spine and genitourinary anomalies with, 181
- AORTA, calcified and dilated ascending, due to atherosclerotic occlusive disease simulating coarctation of aorta; report of case and theory of pathogenesis, 840
- AORTIC aneurysms, abdominal, catheter arteriography in evaluation of, 879
- stenosis, congenital, 800
- stenosis, supraaortic; roentgen analysis of twenty-seven cases, 782
- stenosis, supraaortic, 822
- ARACHNOID CYSTS, primary, of sella turcica, 611
- ARM, swelling of, following radical mastectomy (ab), 521
- ARTERIA TENTORII, internal carotid artery, tentorial branch of; report of three cases, 595
- ARTERIAL calcinosis, familial form of lipocalcigranulomatosis with (ab), 265
- dysplasia of increasing clinical importance; fibromuscular hyperplasia, 907
- ARTERIOGRAPHIC demonstration of compression syndromes of thoracic outlet (ab), 766
- ARTERIOGRAPHY, accuracy of preoperative staging of bladder tumors by: 5-year study (ab), 1023
- and retroperitoneal gas studies, diagnosis of suprarenal mass lesions by, 215
- catheter, in evaluation of abdominal aortic aneurysms, 879
- in circumferential compression edema, 898
- ARTERIOVENOUS malformation of vein of Galen, heart failure in infancy due to; report of case, 653
- malformations, intracranial, skull changes associated with, 525
- shunting in cerebrovascular occlusive disease, 631
- ARTHRITIS, RHEUMATOID, lungs and pleura in, 334
- scintillation scanning in (ab), 523
- ARTHROGRAPHY, knee, value of, in children (ab), 265
- ASTHMA in infancy (ab), 513
- ASTROCYTOMA involving corpus callosum, radiologic detection of, 575



- ATHEROMATOUS OCCLUSIVE DISEASE simulating coarctation of aorta, calcified and dilated ascending aorta due to; report of case and theory of pathogenesis, 840
- ATHEROSCLEROSIS in rabbit visualized by cardioventriculography and subtraction, 844
- ATRIAL SEPTAL DEFECT closure, diversion of inferior vena cava into left atrium following, 851
- AZYGOS anterior cerebral artery (A.C.A.), clinical significance of, 602
- vein, total anomalous pulmonary venous drainage into, 378
- BARIUM enema examinations, further experience with Dulcolax in (ab), 515
- sulfate, acute toxicity of, administered intragastrically (ab), 515
- BILE DUCTS and portal venous system, visualization of (ab), 260
- BILHARZIAL POLYPI of urinary bladder (ab), 261
- BIOLOGY, importance of, in radiotherapy (ab), 1024
- BLADDER tumors, accuracy of preoperative staging of, by arteriography: 5-year study (ab), 1023
- BLOOD PRESSURE changes induced by subclavian injection of methylglucamine diatrizoate (renografin), 914
- BOOK REVIEWS
- The Yearbook of Radiology (1965-1966). Radiologic Diagnosis edited by John F. Holt and Walter M. Whitehouse. Radiation Therapy edited by Howard B. Latourette, 255
- Management of the Patient with Cancer. Edited by Thomas F. Nealon, Jr., 255
- Human Pathology: an Introduction to Medicine. By Robert P. Morehead, 501
- Pneumoencephalography and Cerebral Angiography. By Bernard S. Epstein, 763
- Radiologic Diagnosis in Infants and Children. By Armand E. Brodeur, 1005
- BONE changes in urticaria pigmentosa: report of case (ab), 767
- infarction, fibrosarcoma complicating, in caisson worker: case report (ab), 264
- intra-osseous ganglia of (ab), 262
- lesions, detection of, with strontium-85 scintiscan (ab), 1029
- sclerotic lesions of, in myeloma (ab), 768
- BRACHIOCEPHALIC vascular syndrome associated with cervical rib (ab), 1015
- BRAIN abscess in children; radiologic methods for early recognition, 675
- abscess, possibilities for incorrect diagnosis of, by means of carotid angiography (ab), 769
- lesions, mercury-197 and mercury-203 chlormerodrin for evaluation of, using rectilinear scanner and scintillation camera (ab), 1026
- scanning, comparative study of mercury-197 chlormerodrin and mercury-203 chlormerodrin for (ab), 521
- scanning, isotope, post-angiography; positive or negative?, 672
- BREAST cancer, evaluation of postoperative prophylactic radiation therapy in (ab), 1022
- BRONCHIAL brushing in diagnosis of peripheral lung lesions; preliminary report, 474
- BRONCHOGRAPHIC agent dionosil aqueous, clinical and pathological reactions to, 468
- BRONCHUS, atresia of, to apical-posterior segment of left upper lobe, 350
- occult carcinoma of (ab), 512
- BURNS, third-degree, about foot and ankle, limb-length discrepancy after: report of four cases (ab), 263
- CAMERA, spark-imaging, and conventional scintillation scanner, comparison of clinical results obtained with (ab), 1029
- CANCER, breast, evaluation of postoperative prophylactic radiation therapy in (ab), 1022
- chemotherapy for lung, in intra-aortic infusion (ab), 1030
- lung, measurable proved, in relation to growth rate, survival of men with, 404
- nasopharyngeal, radium source appliance for treatment of (ab), 1024
- of lung, laminagraphic studies of vascular patterns in, with special reference to "circumscribed" variety, 397
- CARBON DIOXIDE insufflation, translumbar retroperitoneal, 212
- CARCINOMA, occult, of bronchus (ab), 512
- of cervix, pelvic angiography and lymphangiography in evaluation of patient with (ab), 1023
- of right side of colon in patients treated for anemia, delay in diagnosis of (ab), 259
- CARDIO-ESOPHAGEAL function in profile (in upright position, with trunk bent and in decubitus position), radiologic examination of (ab), 1018
- CARDIOVASCULAR catheterization, percutaneous, safety guidespring for, 957
- CARDIOVENTRICULOGRAPHY and subtraction, atherosclerosis in rabbit visualized by, 844
- CATHETER arteriography in evaluation of abdominal aortic aneurysms, 879
- position: an aid in diagnosis of dissecting aneurysm of thoracic aorta, 836
- CATHETERIZATION, percutaneous cardiovascular, safety guidespring for, 957
- CAVOGRAPHY following plication of inferior vena cava, 888
- CENTRAL NERVOUS SYSTEM, cysticercosis of, roentgen study of (ab), 766
- CEREBRAL angiography, possibilities for error in (ab), 768
- artery, azygos anterior, (A.C.A.), clinical significance of, 602

- leptomeningeal cysts of developmental origin, 570  
 ventricular-cardiac atrial shunts, roentgenographic manifestations of complications secondary to, 689
- CEREBROVASCULAR occlusive disease, arteriovenous shunting in, 631
- CERVICAL rib, brachiocephalic vascular syndrome associated with (ab), 1015  
 spine, vertebral artery insufficiency secondary to trauma and osteoarthritis of (ab), 1014
- CERVICOCRANIAL circulation, posterior, angiography of, 660
- CERVIX, carcinoma of, pelvic angiography and lymphangiography in evaluation of patient with (ab), 1023
- CESIUM 137, deposition of fallout on forage and transfer to milk (ab), 1031
- CHEMOTHERAPY for lung cancer in intra-aortic infusion (ab), 1030  
 of tuberculosis of spine (ab), 523
- CHEST disease, non-tuberculous, found in mass x-ray survey in Vancouver, B.C. (ab), 1017  
 pulmonary parenchymal findings in blunt trauma to, 343  
 roentgenography, application to impedance pneumograph to; useful medical by-product of space program, 487  
 unit, combination adult-pediatric, 492
- CHILDREN, brain abscess in; radiologic methods for early recognition, 675  
 large dose urography in (ab), 517  
 right middle lobe syndrome in (ab), 1016  
 value of knee arthrography in (ab), 265  
 voiding cystourethrography in; use of rapid film changer, 192
- CHOLANGIO-CHOLECYSTOGRAPHY, peroral, with SH 771: results and side effects in 766 examinations (ab), 1020
- CHOLANGIOGRAPHY, operative (ab), 260  
 oral, in combination with gastrointestinal examination (ab), 1020
- CHROMOSOME studies on women formerly employed as luminous-dial painters (ab), 266
- CLATWORTHY's sign (ab), 514
- CONRAY 60 per cent and vacuum uterine cannula, hysterosalpingography with, 244
- CONTRAST agents, study of 3, in 2,234 intravenous pyelographies, 79  
 medium, increased dosage of, in routine intravenous urography, 77
- CORPUS CALLOSUM, agenesis of, angiographic findings in, 579  
 radiologic detection of astrocytoma involving, 575
- CRANIAL and intracranial lesions in neurofibromatosis, 550  
 collateral pathways in stroke syndrome, 637
- CYSTICERCOSIS of central nervous system, roentgen study of (ab), 766
- CYSTOURETHROGRAPHY, voiding, in children; use of rapid film changer, 192
- CYSTS, arachnoid, primary, of sella turcica, 611  
 cerebral leptomeningeal, of developmental origin, 570  
 traumatic extradural, of spine, 713
- CYTODIAGNOSIS, oral, errors of: report of follow-up of 1,801 patients (ab), 774
- DENTATE ligaments, anatomic, myelographic and cinemyelographic study of, 704
- DIONOSIL AQUEOUS, bronchographic agent, clinical and pathological reactions to, 468
- DIURESIS, high, factor in preventing vesicoureteral reflux (ab), 518
- DIVERTICULA, urethral, in women, 200
- DOUGLAS A. Quick, 1891-1966 (E), 984
- DRIP INFUSION PYELOGRAPHY, effect of, on renal function, 102  
 its evaluation as routine examination, 96
- DUCHENNE muscular dystrophy, increased thickness of fibula in, 744
- DULCOLAX, further experience with, in barium enema examinations (ab), 515
- DYSPROTEINEMIA, idiopathic periosteal hyperostosis with: new clinical entity (ab), 767
- EATON agent pneumonia, roentgen features of, 300
- ECHOCARDIOGRAM: ultrasound technic for detection of pericardial effusion (ab), 258
- EDEMA, circumferential compression, arteriography in, 898
- EDITORIALS  
 The Phenomenon of Extravasation and Its Relation to Acute Urinary Obstruction, 249  
 Martin Schneider, M.D., 1913-1966, 251  
 Electrical Impedance Spirometry, 497  
 The Radiologist's Responsibility for Neuroradiology, 754  
 George W. Grier, M.D., 1882-1966, 757  
 The Sixty-seventh Annual Meeting of the American Roentgen Ray Society, 980  
 Douglas A. Quick, 1891-1966, 984
- ELECTRICAL IMPEDANCE detection, tetrapolar, intrathoracic volumic events by, 482  
 spirometry (E), 497
- EMBOLISM, pulmonary, and pulmonary angiogram, observations on, 859
- EMPHYSEMA, lobar, infantile; report of two cases with unusual roentgenographic manifestation, 354
- ENTEROPATHY, protein-losing: report of two new cases (ab), 514
- EPIPHYSIS, slipped capital femoral, in renal rickets: report of three cases (ab), 263
- ESOPHAGUS, perforations and fistulae in course of radiotherapy of malignant tumors of (ab), 1024
- EXHIBITS, scientific, 987  
 technical, 1000



- FEMORAL arteries, superficial, stationary waves of; report of case occurring in patient with rheumatic heart disease and popliteal arterial emboli, 901
- FIBROMUSCULAR hyperplasia; an arterial dysplasia of increasing clinical importance, 907
- FIBROSARCOMA complicating bone infarction in caisson worker: case report (ab), 264
- FIBROSIS, idiopathic retroperitoneal (ab), 1019
- FIBULA, increased thickness of, in Duchenne muscular dystrophy, 744
- FOOT and ankle, limb-length discrepancy after third-degree burns about: report of four cases (ab), 263
- FURNITURE POLISH ingestion, pulmonary complications following; report of 21 cases, 323
- GALL-STONES, disappearing (ab), 260
- GANGLIA, intra-osseous, of bone (ab), 262
- GAS, retroperitoneal, studies and arteriography, diagnosis of suprarenal mass lesions by, 215
- GASTRIC WALL, thickness of, determined by megavoltage roentgenography (ab), 513
- GASTROINTESTINAL examination, oral cholangiography in combination with (ab), 1020
- GENITOURINARY anomalies, and association of lumbosacral spine, with imperforate anus, 181
- GEORGE W. Grier, M.D., 1882-1966 (E), 757
- GUIDESPRING, safety, for percutaneous cardiovascular catheterization, 957
- HEAD, horizontal laminography of, with ultra-sound (ab), 1014
- HEART disease, congenital, sign of: calcification of pulmonary artery and enlargement of right ventricle; Eisenmenger syndrome—pulmonary hypertension, increased pulmonary resistance, and reversal of blood flow, 369  
failure in infancy due to arteriovenous malformation of vein of Galen; report of case, 653
- HEMATOMA, perirenal, renal infarct with, 70
- HEPARIN, treatment with: wheezing due to pulmonary embolism (ab), 513
- HEPATOMA induced by thorium dioxide (thorotrast) (ab), 1030
- HG<sup>203</sup> CHLORMERODRIN, effect of acute and chronic ureteral occlusion upon renal handling of, 163
- HICKEY LECTURE, 1966; glimpses through pulmonary window; interpretation of radiologic evidence in disorders of lungs, 269
- HIGH ENERGY LASER pulses on primate liver, effect of (ab), 1025
- HIP joint, Paget's disease of (ab), 767
- HODGKIN'S DISEASE, some reflections of treatment and prognosis of (ab), 1022
- HYBRID radioisotope scanner (ab), 1029
- HYDROMYELIA and syringomyelia, roentgenologic findings in, 695
- HYDRONEPHROSIS and hydroureter, nonobstructive, 172
- HYPERPLASIA, fibromuscular; an arterial dysplasia of increasing clinical importance, 907
- HYPERTENSION, portal, from idiopathic superior vena caval obstruction (ab), 770  
renal, pheochromocytoma causing, 222  
renovascular, excretory urography in; minute sequence filming and osmotic diuresis, 114
- HYPOGASTRIC (umbilical) artery, distal, obstruction of lower ureter by, 160
- HYPOPROTEINAEMIA, familial neonatal, with exudative enteropathy and intestinal lymphangiectasis (ab), 258
- HYPOTHYROIDISM, thyroidal <sup>131</sup>I turnover in: correlation with thyrotropin responsiveness (ab), 1026
- HYSTEROSALPINGOGRAPHY, lymphangiogram following, 233  
uterine lymphogram following, 236  
with conray 60 per cent and vacuum uterine cannula, 244
- <sup>131</sup>I human serum albumin, positive delineation of human tumors with (ab), 522
- I<sup>131</sup>, lung scans with, labelled macroaggregated human serum albumin (MAA), 416
- ILIOFEMORAL arteriosclerotic obstruction, "nonsurgical" treatment of (ab), 771
- IATROGENICS (ab), 1025
- INFANCY, asthma in (ab), 513  
heart failure in, due to arteriovenous malformation of vein of Galen; report of case, 653
- INFANTILE lobar emphysema; report of two cases with unusual roentgenographic manifestation, 354
- INFERIOR VENA CAVA, cavography following plication of, 888  
diversion of, into left atrium following atrial septal defect closure, 851
- INFLUENZA VIRUS, experiments with, cytodiagnosis and pathogenesis of lung tumors and: optical and electron microscopic studies (ab), 1018
- INTERNAL CAROTID ARTERY (arteria tentorii), tentorial branch of; report of three cases, 595
- INTESTINAL scleroderma, malabsorption in: correction by antibiotics (ab), 1018
- INTRACRANIAL and cranial lesions in neurofibromatosis, 550  
arteriovenous malformations, skull changes associated with, 525  
lesions, reliability of scintillation scanning for detection of (ab), 1026  
space-occupying lesions, diagnosis of, by association of gamma-encephalography and echo-encephalography (ab), 1014  
structures, dynamic displacements of, simulating mass lesions, 535

- tumors, differentiation of extrinsic from intrinsic, with particular reference to posterior fossa tumors, 542
- INTRATHORACIC volemic events by tetrapolar electrical impedance detection, 482
- INTRAUTERINE TRANSFUSION by multiple-needle technique (ab), 516
- roentgenologic aspects of (ab), 516
- IODINE-131, placental scanning with (ab), 516
- IODIZED OIL, venous intravasation of, during myelography; report of case with roentgenologic evidence of contrast medium in iliac veins and in lungs, 720
- ISOPAQUE 60 per cent and urografin 76 per cent in angiocardiology, osmotic effects of, 922
- ISOTOPE brain scanning, post-angiography; positive or negative?, 672
- KIDNEY, metastatic tumors in (ab), 261
- KLIPEL-TRENAUNAY syndrome, roentgenologic contribution to pathogenesis of (ab), 262
- KNEE arthrography in children, value of (ab), 265
- KYPHOSIS, adolescent (Morbus Scheuermann), contribution to (ab), 264
- LAMINAGRAPHIC studies of vascular patterns in cancer of lung with special reference to "circumscribed" variety, 397
- LAMINOGRAPHY, horizontal, of head with ultra-sound (ab), 1014
- LEPTOMENINGEAL cysts, cerebral of developmental origin, 570
- LEUKEMIA, radiation-related, in Hiroshima and Nagasaki, 1946-1964: I. Distribution, incidence and appearance time (ab), 1031
- LIPOCALCIGRANULOMATOSIS, familial form of, with arterial calcinosis (ab), 265
- LIPOMA, subpleural, roentgen diagnostic observation in, 465
- LIPOMATOSIS, mediastinal, secondary to steroid therapy, 461
- LIPOSARCOMA of extremities and limb girdles: study of two hundred twenty-two cases (ab), 520
- LIVER and pancreas, tomography of, with combined pneumoperitoneum and retroperitoneum (ab), 1019
- primate, effect of high energy laser pulses on (ab), 1025
- scanning, practical applications of (ab), 1028
- LOBAR emphysema, infantile; report of two cases with unusual roentgenographic manifestation, 354
- LUMINOUS-DIAL painters, chromosome studies on women formerly employed as (ab), 266
- LUNG abscess (ab), 1017
- cancer, chemotherapy for, in intra-aortic infusion (ab), 1030
- cancer, measurable proved, in relation to growth rate, survival of men with, 404
- cancer of, laminagraphic studies of vascular patterns in, with special reference to "circumscribed" variety, 397
- disseminated interstitial diseases of (ab), 1015
- lesions, peripheral, bronchial brushing in diagnosis of, 474
- noninfectious necrotizing granulomatosis of; Wegener's syndrome (ab), 1016
- scan, reaction following administration of macroaggregated albumin (MAA) for, 427
- scanning following radioaerosol inhalation (ab), 1027
- scanning—technique and some diagnostic uses (ab), 1027
- scans with  $I^{131}$  labelled macroaggregated human serum albumin (MAA), 416
- tumors, cytodiagnosis and pathogenesis of, and experiments with influenza virus: optical and electron microscopic studies (ab), 1018
- LUNGS and pleura in rheumatoid arthritis, 334
- disorders of, interpretation of radiologic evidence in; glimpses through pulmonary window; Hickey Lecture, 1966, 269
- LYMPHANGIOGRAM following hysterosalpingography, 233
- LYMPHANGIOGRAPHY, improved, simple technique of, 948
- improved technique of, 952
- LYMPHOGRAM, uterine, following hysterosalpingography, 236
- complications encountered in 522 examinations (ab), 772
- complications of (ab), 771
- errors in (ab), 769
- in patient with nonreactive tuberculosis, 436
- pulmonary oil deposition in patients subjected to: detection by thoracic photoscan and sputum examination (ab), 1017
- MACROAGGREGATED albumin (MAA), reaction following administration of, for lung scan, 427
- human serum albumin (MAA), lung scans with  $I^{131}$  labelled, 416
- MARTIN Schneider, M.D., 1913-1966 (E), 251
- MASTECTOMY, radical, swelling of arm following (ab), 521
- MEGACOLON in adults (ab), 514
- MEGASIGMOID syndrome in psychotic patients (ab), 259
- MENINGO-HYPOPHYSEAL trunk, roentgenology of, 583
- MERCURY-197 and mercury-203 chlormerodrin for evaluation of brain lesions using rectilinear scanner and scintillation camera (ab), 1026
- chlormerodrin and mercury-203 chlormerodrin, comparative study of, for brain scanning (ab), 521
- MERISOPROL Hg 197, splenic scintiscans with: new radioactive pharmaceutical agent (ab), 522
- METHYLGLUCAMINE diatrizoate (renografin), subclavian injection of, blood pressure changes



- induced by, 914
- METHYSERGIDE therapy, fibrotic disorders associated with, for headache, 259
- MORBUS SCHEUERMANN, contribution to adolescent kyphosis (ab), 264
- MUSCULAR DYSTROPHY, Duchenne, increased thickness of fibula in, 744
- MYELOGRAPHY, venous intravasation of iodized oil during; report of case with roentgenologic evidence of contrast medium in iliac veins and lungs, 720
- MYELOMA, sclerotic lesions of bone in (ab), 768
- NASOPHARYNGEAL cancer, radium source appliance for treatment of (ab), 1024
- NEPHROTOGRAPHY, value of, in differential diagnosis of abdominal masses, 935
- NEUROFIBROMATOSIS, 557  
cranial and intracranial lesions in, 550
- NEUROPATHY, peripheral, radiation-induced (ab), 766
- NEURORADIOLOGY, optic canal in (ab), 510  
radiologists' responsibility for (E), 754
- NEWBORN, asphyxiating thoracic dystrophy of, 358
- OPTIC CANAL in neuroradiology (ab), 510
- ORTHODIAGNOSTIC shift pelvimetry (ab), 515
- OSTEOPOROSIS, scurvy, and siderosis in Johannesburg Bantu (ab), 263
- PAGET's disease of hip joint (ab), 767
- PAGET-VON SCHRÖTTER syndrome; angiographic features of primary venous obstruction of upper extremity, 388
- PAROTID masses, sialography in differential diagnosis of (ab), 511
- PEGOT-CRUEILHIER-BAUMGARTEN syndrome (ab), 770
- PELVIC angiography and lymphangiography in evaluation of patient with carcinoma of cervix (ab), 1023
- PELVIMETRY, orthodiagnostic shift (ab), 515
- PERICARDIAL EFFUSION, angiocardiographic diagnosis of, 874  
ultrasound technic for detection of: echocardiogram (ab), 258
- PERIOSTEAL hyperostosis, idiopathic, with dysproteïnemia: new clinical entity (ab), 767
- PERIPELVIC extravasation during intravenous urography, 41
- PHARYNGEAL-vertebral anastomosis, ascending, 599
- PHENACETIN overdose, radiologic observations associated with (ab), 519
- PHEOCHROMOCYTOMA, angiography of, 225  
causing renal hypertension, 222
- PHOTOFLUOROGRAPHY installation, battery-operated, mounted in vehicle—initial trial (ab), 265
- PHOTOGRAPHIC spot films and radiographic spot films, 961
- PHOTOSCANNING, radioisotope, in pulmonary disease (ab), 521
- PITUITARY adenomas, extrasellar extension of; clinical and neuroradiological considerations, 616  
tumors, symposium on: I. clinical management and results of treatment of chromophobe pituitary adenoma (ab), 1020  
tumors, symposium on: II. radiology in diagnosis and management (ab), 1021  
tumors, symposium on: III. historical aspects of radiotherapy, present treatment technique and results (ab), 1021  
tumors, symposium on: IV. unusual pathological features (ab), 1022
- PLACENTA, opacification of amniotic fluid for localization of (ab), 516
- PLACENTAL scanning with iodine-131 (ab), 516
- PLASMA CELL pneumonia; Pneumocystis Carinii pneumonia; roentgenographic, pathologic and clinical correlation, 318
- PLEURAL calcification as sign of silicosis (ab), 258
- PNEUMOCYSTIS CARINII pneumonia (plasma cell pneumonia); roentgenographic, pathologic and clinical correlation, 318
- PNEUMOENCEPHALOGRAPHY, upright planigrams in, 749
- PNEUMOGRAPH, impedance, application of, to chest roentgenography; useful medical by-product of space program, 487
- PNEUMOGRAPHY, orbital (ab), 510
- PNEUMOMEDIASTINOGRAPHY, 451
- PNEUMONIA, Eaton agent, roentgen features of, 300  
Pneumocystis Carinii (plasma cell pneumonia); roentgenographic, pathologic and clinical correlation, 318  
varicella, roentgenographic manifestations of, with postmortem correlation, 305
- POLYCYTHEMIA rubra vera and radioactive phosphorus—90 patients (ab), 1030
- PORTAL VENOUS system, and bile ducts, visualization of (ab), 260
- POSTERIOR fossa tumors, differentiation of extrinsic from intrinsic intracranial tumors with particular reference to, 542  
pituitary extract, angio-renographic studies in oestrone-pretreated adrenalectomized and hypophysectomized rats following administration of (ab), 520
- PRESIDENT'S ADDRESS; problems in radiology of today and future, 777
- PROSTATE and urinary bladder floor veins, roentgenographic exploration of; periprostic phlebography: new method, 208
- PROTEIN-losing enteropathy: report of two new cases (ab), 514
- PSEUDOSTENOSIS of renal artery, 143
- PSEUDOVASCULAR ring resulting from right lung agenesis, normal aortic arch, and patent ductus arteriosus; case report, 365
- PSYCHOTIC patients, megasigmoid syndrome in (ab), 259

- PULMONARY artery, calcification of, and enlargement of right ventricle; sign of congenital heart disease; Eisenmenger syndrome—pulmonary hypertension, increased pulmonary resistance, and reversal of blood flow, 369  
 complications following furniture polish ingestion; report of 21 cases, 323  
 disease, radioisotope photoscanning in (ab), 521  
 edema, acute interstitial, 291  
 edema, lateral roentgenogram in, 279  
 embolism, amniotic, nonfatal, 434  
 embolism and pulmonary angiogram, observations on, 859  
 embolism, wheezing due to: treatment with heparin (ab), 513  
 histoplasmosis, roentgen diagnosis of (ab), 512  
 manifestations of tuberous sclerosis, 734  
 oil deposition in patients subjected to lymphography: detection by thoracic photoscanner and sputum examination (ab), 1017  
 parenchymal findings in blunt trauma to chest, 343  
 venous drainage, total anomalous, into azygos vein, 378  
 window, glimpses through; interpretation of radiologic evidence in disorders of lungs; Hickey Lecture, 1966, 269
- PYELOGRAM, intravenous, changes on, in renal artery disease, 107  
 urea washout test, normal; study of 33 normotensive controls, 88
- PYELOGRAPHIES, intravenous, study of 3 contrast agents in 2,234, 79
- PYELOGRAPHY, drip infusion, effect of, on renal function, 102  
 drip infusion; its evaluation as routine examination, 96  
 infusion, in renal insufficiency (ab), 517
- QUICK, Douglas A., 1891-1966 (E), 984
- RADIATION-induced peripheral neuropathy (ab), 766  
 ionizing, detection of effects of, by population studies (ab), 265
- RADIOACTIVE phosphorus and polycythemia rubra vera—90 patients (ab), 1030
- RADIOAEROSOL inhalation, lung scanning following (ab), 1027
- RADIOGRAPHIC spot films and photographic spot films, 961
- RADIOGRAPHY, colored, investigations in, 966
- RADIOISOTOPE photoscanning in pulmonary disease (ab), 521  
 scanner, hybrid (ab), 1029  
 scanning, quantification of respiratory motion artifact in, with rectilinear focused collimator scanner and gamma scintillation camera (ab), 1029
- RADIOLOGIC detection of astrocytoma involving corpus callosum, 575  
 evidence, interpretation of, in disorders of lungs; glimpses through pulmonary window; Hickey Lecture, 1966, 269  
 examination of cardio-esophageal junction in profile (in upright position, with trunk bent and in decubitus position) (ab), 1018  
 methods for early recognition; brain abscess in children, 675  
 observations associated with phenacetin overdosage (ab), 519
- RADIOLOGIST's responsibility for neuroradiology (E), 754
- RADIOLOGY, critical assessment of importance of, in investigation of renal vascular hypertension (ab), 517  
 of today and future, problems in; President's address, 777
- RADIOTHERAPY, importance of biology in (ab), 1024  
 of malignant tumors of esophagus, perforation and fistulae in course of (ab), 1024
- RADIUM source appliance for treatment of nasopharyngeal cancer (ab), 1024
- RENAL agenesis and dysgenesis, angiography of, 137  
 angiography, subtraction technique in (ab), 773  
 artery aneurysms, 132  
 artery disease, changes on intravenous pyelogram in, 107  
 artery, pseudostenosis of, 143  
 colic causing perinephric abscess, spontaneous urinary extravasation associated with, 47  
 colic, urography during (ab), 261  
 extravasation, spontaneous, during intravenous urography, 27  
 function, effect of drip infusion pyelography on, 102  
 handling of Hg<sup>203</sup> chlormerodrin, effect of acute and chronic ureteral occlusion upon, 163  
 hypertension, pheochromocytoma causing, 222  
 infarct with perirenal hematoma, 70  
 infarctions, subtotal, 63  
 insufficiency, infusion pyelography in (ab), 517  
 lesion of tuberous sclerosis, roentgenographic findings in, 739  
 pelvis, spontaneous rupture of, 54  
 rickets, slipped capital femoral epiphysis in: report of three cases (ab), 263  
 trauma, roentgenologic evaluation of, with emphasis on renal angiography, 1  
 vascular hypertension, critical assessment of importance of radiology in investigation of (ab), 517  
 vascularity in dogs, effects of angiotensin on, 927  
 vein thrombosis, roentgen diagnosis of; clinical aspects, 122
- RENOVASCULAR hypertension, excretory urography in; minute sequence filming and osmotic diuresis, 114
- RHABDOMYOSARCOMA in children, pathology of (ab), 1026

- RHEUMATOID ARTHRITIS, lungs and pleura in, 334  
scintillation scanning in (ab), 523
- RIGHT MIDDLE LOBE syndrome in children (ab), 1016
- ROENTGEN diagnosis of pulmonary histoplasmosis (ab), 512  
diagnosis of renal vein thrombosis; clinical aspects, 122  
diagnostic observation in subpleural lipoma, 465  
features of Eaton agent pneumonia, 300  
study of cysticercosis of central nervous system (ab), 766
- ROENTGENOGRAM, lateral, in pulmonary edema, 279
- ROENTGENOGRAPHIC exploration of prostate and urinary bladder floor veins; periprostic phlebography: new method, 208  
findings in renal lesion of tuberous sclerosis, 739  
manifestation, unusual, report of two cases with; infantile lobar emphysema, 354  
manifestations of complications secondary to cerebral ventricular-cardiac atrial shunts, 689  
manifestations of varicella pneumonia with post-mortem correlation, 305
- ROENTGENOGRAPHY, chest, application of impedance pneumograph to; useful medical by-product of space program, 487  
megavoltage, thickness of gastric wall determined by (ab), 513
- ROENTGENOLOGIC aspects of intrauterine transfusion (ab), 516  
changes in uterine isthmus insufficiency, 239  
contribution to pathogenesis of Klippel-Trenaunay syndrome (ab), 262  
evaluation of renal trauma with emphasis on renal angiography, 1  
findings in syringomyelia and hydromyelia, 695
- ROENTGENOLOGY of meningo-hypophyseal trunk, 583
- SCHNEIDER, Martin, M.D., 1913-1966 (E), 251
- SCIENTIFIC exhibits, 987
- SCURVY, osteoporosis, and siderosis in Johannesburg Bantu (ab), 263
- SELENITE (74 Se) as tumor-localizing agent in man (ab), 1028
- SELLA TURCICA, changes of, in chromophobic adenomas and eosinophilic adenomas (ab), 510  
primary arachnoid cysts of, 611
- SIALOGRAPHY in differential diagnosis of parotid masses (ab), 511
- SIDEROSIS, scurvy, and osteoporosis in Johannesburg Bantu (ab), 263
- SILICATOSIS, pleural calcification as sign of (ab), 258
- SINUS of Valsalva aneurysms (ab), 258
- SIXTY-SEVENTH annual meeting of American Roentgen Ray Society (E), 980
- SKULL changes associated with intracranial arteriovenous malformations, 525
- SPARK-IMAGING CAMERA and conventional scintillation scanner, comparison of clinical results obtained with (ab), 1029
- SPINE, lumbosacral, and genitourinary anomalies with imperforate anus, association of, 181  
traumatic extradural cysts of, 713  
tuberculosis of, chemotherapy of (ab), 523
- SPIROMETRY, electrical impedance (E), 497
- SPLENIC scintiscans with merisoprol Hg 197; new radioactive pharmaceutical agent (ab), 522
- SPLENOPORTOGRAPHY, possibilities for incorrect diagnosis in (ab), 769
- SPOT FILMS, photographic, and radiographic spot films, 961
- STATIONARY WAVES of superficial femoral arteries; report of case occurring in patient with rheumatic heart disease and popliteal arterial emboli, 901
- STEROID therapy, mediastinal lipomatosis secondary to, 461
- STROKE, aneurysm as cause of, 647  
syndrome, cranial collateral pathways in, 637
- STRONTIUM-85 scintiscan, detection of bone lesions with (ab), 1029
- SUBCLAVIAN ARTERY, occlusive disease of (ab), 770
- SUBTRACTION technic: video and color methods (ab), 773  
technique in renal angiography (ab), 773
- SUPERIOR VENA CAVAL obstruction, idiopathic, portal hypertension from (ab), 770  
syndrome due to tuberculosis; report of three cases, 440
- SUPRARENAL mass lesions, diagnosis of, by retroperitoneal gas studies and arteriography, 215
- SUPRAVALVAR aortic stenosis; roentgen analysis of twenty-seven cases, 782
- SUPRAVALVULAR aortic stenosis, 822
- SYRINGOMYELIA and hydromyelia, roentgenologic findings in, 695
- TECHNICAL exhibits, 1000
- TETRAPOLAR electrical impedance detection, intrathoracic volumic events by, 482
- THORACIC aorta, an aid in diagnosis of dissecting aneurysm of: catheter position, 836  
dystrophy, asphyxiating, of newborn, 358  
outlet, arteriographic demonstration of compression syndromes of (ab), 766
- THYROID nodules as a late sequela of radioactive fallout in a Marshall island population exposed in 1954 (ab), 1031
- THYROIDAL <sup>131</sup>I turnover in hypothyroidism: correlation with thyrotropin responsiveness (ab), 1026
- TOMOGRAPHIC exploration of abdominal viscera by combined pneumoperitoneum and retro-pneumoperitoneum (ab), 1019
- TOMOGRAPHY of liver and pancreas with combined pneumoperitoneum and retro-pneumoperitoneum (ab), 1019
- TRANSLUMBAR retroperitoneal carbon dioxide insufflation, 212



- TUBERCULOSIS, nonreactive, lymphography in patient, with 436  
of spine, chemotherapy of (ab), 523  
superior vena caval syndrome due to; report of three cases, 440
- TUBEROUS SCLEROSIS, angiographic findings in patient with, 723  
pulmonary manifestations of, 734  
renal lesion of, roentgenographic findings in, 739
- TURANO'S method applied to study of motor-functions of hollow muscular viscera (simultaneous roentgen cinematographic and pressure recording) (ab), 772
- ULTRA-SOUND, horizontal laminography of head with (ab), 1014
- URETER, lower, obstruction of, by distal hypogastric (umbilical) artery, 160
- URETERAL occlusion, effect of acute and chronic, upon renal handling of  $\text{Hg}^{203}$  chlormerodrin, 163  
pacemaker, use of, in treatment of ureteral reflux (ab), 519
- Ureteropelvic junction obstruction, 148
- URETHRAL diverticula in women, 200
- URINARY bladder, bilharzial polypi of (ab), 261  
extravasation, spontaneous, associated with renal colic causing perinephric abscess, 47  
obstruction, acute, phenomenon of extravasation and its relation to (E), 249
- UROGRAFIN 76 per cent and isopaque 60 per cent in angiocardiology, osmotic effects of, 922
- UROGRAPHY during renal colic (ab), 261  
excretory, in renovascular hypertension; minute sequence filming and osmotic diuresis, 114  
intravenous, peripelvic extravasation during, 41  
intravenous, spontaneous renal extravasation during, 27  
large dose, in children (ab), 517  
routine intravenous, increased dosage of contrast medium in, 77
- URTICARIA PIGMENTOSA, bone changes in: report of case (ab), 767
- UTERINE isthmus insufficiency, roentgenologic changes in, 239  
lymphogram following hysterosalpingography, 236
- VARICELLA pneumonia, roentgenographic manifestations of, with postmortem correlation, 305
- VEIN OF GALEN, arteriovenous malformation of, heart failure in infancy due to; report of case, 653
- VENTRICLE, right, enlargement of, calcification of pulmonary artery and: sign of congenital heart disease; Eisenmenger syndrome—pulmonary hypertension, increased pulmonary resistance, and reversal of blood flow, 369
- VERTEBRAL artery insufficiency secondary to trauma and osteoarthritis of cervical spine (ab), 1014
- VESICoureTERAL reflux, factor in preventing, high diuresis (ab), 518
- VOLEMIC events, intrathoracic, by tetrapolar electrical impedance detection, 482
- WEGENER'S syndrome: noninfectious necrotizing granulomatosis of lung (ab), 1016
- WHEEZING due to pulmonary embolism; treatment with heparin (ab), 513
- X-RAY image intensification, application of, to orthopaedic surgery (ab). 773



# AUTHOR INDEX TO VOLUME 98

\*=Original Article

- Abel, Miriam, 515  
 Abrahams, C., 263  
 Abrams, Herbert L., \*822  
 Akerman, M., 1014  
 Aldridge, George, \*102  
 Amplatz, Kurt, \*836  
 Anderson, E. Everett, \*212  
 Anderson, Fred W., 259  
 Anderson, Gerald G., 516  
 Andrews, John T., 766  
 Assadourian, R., 1019
- Babini, L., 1024  
 Baker, David, H., \*181  
 Barden, Robert P., \*269  
 Barry, William F., Jr., \*132  
 Basch, Richard I., \*54  
 Bean, H. A., 1017  
 Béboux, J.-M., \*436  
 Becker, Joshua A., \*96  
 Bélanger, Raymond, \*70  
 Belli, M., 265  
 Benedek, Thomas G., 767  
 Berdon, Walter E., \*181  
 Berghaus, Horst, \*208  
 Berk, J. Edward, 260  
 Bertan, Vural, \*570  
 Bienstock, Milton, \*200  
 Billmeyer, Fred W., Jr., \*966  
 Bittermann, Wilhelm, \*27  
 Bizzozero, O. J., Jr., 1031  
 Björk, Lars, \*922  
 Blazek, James V., \*888  
 Bloomfield, J. A., 517  
 Bluth, I., \*397  
 Boijesen, Erik, \*225, \*447  
 Bonk, Johan P., \*54  
 Bothwell, T. H., 263  
 Boucot, Katharine R., \*404  
 Boulter, T. R., \*583  
 Boyd, Eldon M., 515  
 Boyd, J. T., 266  
 Braun, William T., \*41  
 Brenner, H., 768  
 Bridges, B., \*723  
 Brodeur, A. E., 519  
 Brown, B. St. John, 767  
 Brown, Donald W., \*416  
 Brown, W. M. Court, 266  
 Bruhn, Fred, 258  
 Brünner, S., \*239  
 Bruns, W. L., 516  
 Bryant, Lester R., 770  
 Brylski, J. R., \*672  
 Buchtala, V., 1020
- Buehl, Isabelle, \*354  
 Bull, Frances E., \*427  
 Bull, J. W. D., \*575  
 Buschke, F., 1022  
 Bussat, Ph. L., \*436
- Cade, Stanford, 1025  
 Caine, Marco, \*27  
 Callow, Allan D., \*879  
 Campbell, John A., \*782  
 Carson, Merl J., \*305  
 Carter, W. F., 516  
 Carter, William J., \*734  
 Catalano, Dino, \*244  
 Cavalieri, Ralph R., 1028  
 Chambers, Donaki E., \*63  
 Charlton, R. W., 263  
 Charms, Bernard L., \*859  
 Charron, Jules, \*70  
 Cheris, David N., \*54  
 Chernak, E. S., 774  
 Chu, Florence C. H., 1022  
 Chynn, Kuo-York, \*653  
 Cioeco, A., 1031  
 Clark, Richard L., \*888  
 Cohen, William N., \*689  
 Collard, M., 265  
 Conard, R. A., 1031  
 Conrady, Jutta, 1023  
 Constantine, H., 1016  
 Cooley, Robert N., 251  
 Cooper, David A., \*404  
 Cooper, R. D., Jr., 516  
 Cornell, Steven H., \*143  
 Coventry, Mark B., 520  
 Crabbe, W. A., 262  
 Cramer, Glen G., \*836  
 Crosett, Alexander D., Jr., \*739  
 Crosby, E. H., 1017  
 Crummy, Andrew B., \*122  
 Cuevas, C., \*535  
 Curry, George C., \*350  
 Curry, J. L., \*898  
 Curry, Thomas S., III, \*350  
 Cushing, Frederick R., 249  
 Custer, R. Philip, 522
- Darling, Donald B., \*492  
 Daves, Marvin L., 514  
 Dávid, M. A., 520  
 Davidson, Kendrick C., \*550  
 Davis, Thomas P., 1029  
 Dearman, James R., 1025  
 Debonnière, C., 770  
 Dees, Susan C., 1016
- Delgado, Sergio, 263  
 DeNardo, Gerald L., \*416, 1029  
 deParedes, Ramon G., \*1  
 de Roo, T., \*948  
 Desforges, G., 1016  
 Des Rosiers, Raymond J., \*137  
 de Villiers, J. C., 1015  
 Dexter, D., 1017  
 Dilenge, D., 510  
 Ditchek, Theodore, \*236  
 Dolan, Patrick A., 772  
 Don, Conway, \*107  
 Dorfman, Howard D., 264  
 Dotter, Charles T., 771, \*957  
 Douglass, Clifton F., Jr., 517  
 Doust, B. D., 1027  
 Drake, Arnold, 514  
 Duggan, H. E., 1030  
 DuShane, James W., \*800  
 Dutz, Werner, \*318  
 Dworkin, Howard J., \*427
- Edelstein, Josef, \*859  
 Eggum, P. R., 513  
 Egidio, Paul T., 259  
 Eichling, John, 521  
 Einstein, Robert A. J., 511  
 Eisen, A., 767  
 Ekman, H., 518  
 El-Badawi, A. A., 261  
 Elkin, Milton, \*1, \*927, 1023  
 Emmanuel, I. G., 1021  
 Epstein, Bernard S., \*704  
 Etter, Lewis E., 757
- Farrow, Joseph H., 1022  
 Fass, Leroy, 259  
 Felson, Benjamin, 1015  
 Fennessy, John J., \*474  
 Ferguson, Thomas B., \*851  
 Ferris, Ernest J., \*631  
 Fields, William S., \*660  
 Finby, Nathaniel, \*200, 259, \*935  
 Fischman, J. L., \*102  
 Fisher, Mary Stuart, \*233  
 Flavell, Geoffrey, 1017  
 Follis, G., 512  
 Fonkalsrud, Eric W., 770  
 Forsaith, A. L., 1029  
 Fowler, R. S., \*378  
 Fracchia, Alfred A., 1022  
 Franken, E. A., Jr., \*354  
 Frantz, Charles H., 263  
 Freckman, Herman A., 1030

- Friedman, Barry, 523  
 Frielingsdorf, D., 769  
 Frische, Louis H., 771, \*957  
 Fry, Harry L., 1030  
 Gaensler, E. A., 1016  
 Galligioni, Francesco, \*720  
 Ganson, Jorge, 773  
 Gardiner, J. H., 519  
 Gardner, A. M. N., 260  
 Garreta, L., 770  
 Garusi, G. F., 517  
 Gerald, Barry E., \*734  
 Gilson, J. Michael, 1026  
 Girdany, Bertram R., \*961  
 Gleason, D. C., \*279  
 Glenn, James F., \*212  
 Glenn, James S., 259  
 Goldbloom, Richard B., 767  
 Gooding, Charles A., \*602  
 Gottschalk, Alexander, 1029  
 Gould, Howard R., \*192  
 Graham, John R., 259  
 Gramiak, Raymond, \*465  
 Granger, Donald P., 1014  
 Green, Allan E., Jr., 1024  
 Greenberg, A., 1027  
 Greenberg, Wayne V., 1026  
 Greene, Laurence F., \*114  
 Greitz, Torgny V. B., 510  
 Grossman, Herman, \*181  
 Guen, C., 1019  
 Guen, Cl., 1019  
 Guiot, G., 1014  
 Gup, A. K., \*102  
 Guzzo, Francis, \*557  
 Hagstrom, Jack W. C., \*653  
 Hanafée, William, \*647  
 Hancock, James A., Jr., 1024  
 Handa, Hajime, \*595  
 Handa, Jyoji, \*595, \*914  
 Harper, Paul V., 1029  
 Harris, Vivian J., \*744  
 Harris, Willard S., \*744  
 Harrow, Benedict R., \*47  
 Hasting-James, R., 510  
 Head, Louis R., 521  
 Hebert, Donald H., \*300  
 Heckman, Bernard A., \*874  
 Heinz, E. R., \*672  
 Heitzman, E. Robert, \*291  
 Hems, G., 265  
 Herbig, F., 519  
 Hermann, George, \*27  
 Hermann, George, III, 522  
 Hermann, Robert E., 260  
 Herron, Charles S., \*888  
 Hidalgo, John U., 523  
 Hill, B. J., \*637  
 Hipona, Florencio A., \*122, \*236  
 Hiraki, Tatsunosuke, 522  
 Hisada, Kin-ichi, 522  
 Hochberg, Bernard, \*181  
 Hodges, Clarence V., \*222  
 Holden, W. S., 260  
 Holman, Colin B., 754  
 Holmes, R. Brian, 258  
 Holta, Anne Lise, 1019  
 Horwitz, N. H., 1029  
 Howieson, John, \*575  
 Howland, W. J., \*898  
 Hoye, Robert C., 1025  
 Hughes, J. Howell, 521  
 Hunt, James C., \*114  
 Iraci, Giorgio, \*720  
 Irvine, W. J., \*451  
 Izenstark, J. L., 516, \*672  
 Jackson, Reginald J. A., 771  
 Jacobson, B., 518  
 Jannetta, Peter J., \*647  
 Jefferson, A., 1020  
 Jeffries, Graham H., 1018  
 Jimenez, John P., \*323  
 Jimenez, Feliciano F., 1029  
 Jing, Bao-Shan, \*952  
 Johnson, J. E., 1031  
 Johnson, K. G., 1031  
 Johnston, Gerald S., \*163  
 Jongmans, H., 773  
 Judkins, Melvin P., \*225, 771, \*957  
 Kahn, Ivan J., 1018  
 Kahn, Paul C., \*879  
 Kanick, Virginia, \*935  
 Kanoui, Fernand, 1018  
 Katz, Lawrence, \*966  
 Kaufman, B., 774  
 Kaye, Josse, 515  
 Keggi, Kristaps J., 1014  
 Kelly, Prue, \*77  
 Khodadad, Ethylyn Jennings, \*318  
 Kim, Seuk Ky, \*132  
 Kincaid, Owings W., \*800  
 Kircher, Konrad F., \*434  
 Kittredge, Richard D., \*200, \*935  
 Klein, P., 770  
 Kleinfeld, Morris, 258  
 Kock, N. G., 518  
 Kocsis, J., 520  
 Koerner, Herbert J., \*461, \*465  
 Koslowski, L., 773  
 Kovács, K., 520  
 Kraft, Ernest, 259  
 Krueger, Erich G., \*616  
 Kune, G. A., 514  
 Kupic, Edward A., \*822  
 Kurlander, Gerald J., \*782  
 Lang, Erich K., \*215, 1023  
 Langley, G. R., 768  
 Larsen, John Ludv., \*579  
 Larson, Sanford J., \*637  
 László, F. A., 520  
 Laubenberger, Theodor, \*208  
 Lawrence, Philip S., \*79  
 Lazar, S. J., 515  
 LeCompte, Philip R., 259  
 Ledoux-Lebard, Guy, 1018  
 Lee, Fred A., \*961  
 Leger, Lucien, 1018  
 Legré, J., 1019  
 Lehman, J. Stauffer, Jr., \*653  
 LeMay, Marjorie, \*602  
 Lescher, Alfred J., \*637  
 Lester, Richard G., \*323  
 Leucutia, Traian, 980  
 Lewtas, N. A., 1021  
 Light, John P., \*468  
 Linde, Leonard M., 770  
 Lofstrom, J. E., 1029  
 Loken, Merle K., 1026  
 Longmire, William P., Jr., 770  
 Love, Leon, \*137, \*637  
 Lucas, John C., Jr., 1022  
 Lurie, Paul R., \*782  
 Lynch, S. R., 263  
 MacKay, J. S., 1030  
 McAlister, William H., \*160  
 McCallum, Donald, \*215  
 McCormick, Wm. F., \*583  
 McKnight, William B., 1025  
 McRae, D. L., 1014  
 McRae, Donald L., \*695  
 McRae, J., 1027  
 McSheffrey, J. B., 767  
 Macht, Stanley H., \*79  
 Machtey, Israel, 767  
 Mackenzie, C. J. G., 1017  
 Makow, M., 1014  
 Malkin, C., 263  
 Mankin, Henry J., 263  
 Marchal, Maria Theresa, \*482  
 Marchal, Maurice, \*482  
 Margulis, Alexander R., 513  
 Marshall, T. R., 512  
 Martone, Ronald J., 1029  
 Mathisen, Willy, 1019  
 Matson, D. D., \*675  
 Maurer, Elmer R., 1030  
 Maurer, H.-J., 769  
 Maus, J. H., 984  
 Maxfield, William S., 523  
 Mead, Philip B., 516  
 Melick, W. F., 519  
 Mendez, Fernando L., Jr., 1030



- Meng, Chien-Hsing, \*1, \*927  
 Mertz, John, \*215  
 Mertz, John H. O., 1023  
 Meszaros, William T., \*557  
 Metzger, J., 510  
 Mueller, Robert, 771  
 Meyer, John S., \*914  
 Milledge, Robert D., \*734  
 Milne, E. N. C., 515  
 Minton, John P., 1025  
 Moës, C. A. F., 265, \*378  
 Monks, P. J. W., 260  
 Morgan, Trefor, 767  
 Morgan, William Keith C., \*334  
 Morris, J. G., 1027  
 Morrow, Robert M., 1024  
 Moss, Arthur J., 258  
 Mullins, Fitzhugh, 1025  
 Munn, J. D., 265  
 Munster, W., 262  
 Murison, Paul J., 523  
 Murphy, Gerald P., \*163  
  
 Naryka, J. J., 519  
 Neuhauser, E. B. D., \*358, \*675  
 Newsam, J. E., 261  
 Newton, Thomas H., \*907  
 Nierling, Dean A., \*599  
 Nori, Alessandro, \*720  
 Norman, Alex, 264  
 Nourse, Myron, \*215  
 Nourse, Myron H., 1023  
 Nyboer, Jan, \*482, 497  
  
 Ohba, Satoru, 522  
 Olinger, Charles P., \*844  
 O'Loughlin, Bernard J., \*487  
 O'Neill, Francis Edw., 517  
 Ong, Helen, 514  
 Oster, Walter F., \*468  
 Ouimet-Oliva, Denise, \*70  
  
 Palubinskas, A. J., \*907  
 Parfitt, A. M., 258  
 Parker, Brent M., \*851  
 Patel, A. R., 521  
 Patterson, Marcel, 514  
 Pearson, F. G., 512  
 Pearson, O. H., 774  
 Perloff, Dorothee, \*907  
 Perotti, B., 265  
 Petasnick, Jerry P., 1029  
 Peterson, Carl G., Jr., \*192  
 Petite, J., \*436  
 Petry, Eugene L., \*782  
 Picard, Claude, 1018  
 Pietri, H., 1019  
 Pirker, E., 262  
 Pirnar, Tugrul, \*358  
 Poe, N. D., 1027  
  
 Politano, V., \*723  
 Porstmann, W., 262  
 Posnikoff, Jack, 1014  
 Potts, D. Gordon, \*525  
 Pribram, H. F. W., \*542, \*583  
 Prins, Harvey R., \*966  
  
 Queenan, John T., 516  
 Quinn, James L. III, 521  
  
 Raimondi, A. J., \*637  
 Rall, R. E., 1031  
 Ravel, R., \*723  
 Reilly, Emmett D., \*305  
 Reinhold, H., 264  
 Remmers, August R., \*88  
 Reszel, Paul A., 520  
 Reuter, Stewart R., \*447  
 Rhoton, Albert L., Jr., 521  
 Richardson, P., 1017  
 Rigby, C. C., 1026  
 Ring, B. Albert, \*611  
 Robbins, Guy F., 1022  
 Roberts, Gomer W., \*749  
 Roberts, William D., \*434  
 Robertson, Thomas L., \*487  
 Rodnan, Gerald P., 767  
 Romney, Seymour L., 1023  
 Rosenbaum, Harold D., \*77  
 Rosenberg, J. C., 766  
 Ross, R. C., 1030  
 Ross, Ronald J., 510  
 Ross, Steven E., \*961  
 Rowley, John C., 258  
 Ruberti, Renato, \*720  
 Rudd, Kenneth D., 1024  
 Rumbaugh, Calvin L., \*525  
 Russell, Warren M., 513  
 Rüttiman, A., 769  
  
 Saadeh, G. B., 513  
 Sabeen, H. B., 768  
 Sadowsky, Norman L., 259  
 Sairenji, Eiko, 1028  
 Sakamoto, Ko, \*914  
 Salomoni, Enzo, 772  
 Samaan, N., 774  
 Sandler, Henry C., 774  
 Santfilippo, Louis J., 1023  
 Santín, Guillermo, 766  
 Santulli, Thomas V., \*181  
 Sargent, E. Nicholas, \*305, \*487,  
 1014  
 Sarles, H., 1019  
 Sarles, Harry E., \*88  
 Sarles, J.-Cl., 1019  
 Sassi, P., 1024  
 Schechter, M. M., \*713  
 Schechter, Mannie M., \*535  
 Schirmeisen, P., 1020  
  
 Schlegel, J. U., \*102  
 Schmaman, A., 263  
 Schmukler, Morton, 1026  
 Schorsch, Hildegard, \*557  
 Schreiber, Melvyn H., \*88  
 Schwartz, Armin, \*27  
 Scott, Kenneth G., 1028  
 Seftel, H. C., 263  
 Shahinfar, A. H., \*713  
 Shapiro, Jerome H., 516, \*631  
 Shea, Darryl, 263  
 Shealy, C. N., 774  
 Shehadi, William H., 1028  
 Sheps, Sheldon G., \*114  
 Shopfner, Charles E., \*148, \*172  
 Simeone, Frederick A., \*631  
 Simon, J., 510  
 Sirtori, C., 1018  
 Sleisenger, Marvin H., 1018  
 Smitananda, N., 1027  
 Smith, Garland H., \*88  
 Smith, Irvine, 261  
 Smith, Josef R., \*427  
 Solomon, A., 515  
 Sorger, K., 768  
 Soule, Edward H., 520  
 Southwick, Wayne O., 1014  
 Sövényi, E., 520  
 Spencer, Frank C., 770  
 Spira, Jacob, 516  
 Spock, Alexander, 1016  
 Staab, Edward V., 1026  
 Standen, James, \*695  
 Staple, Tom W., \*851  
 Stein, Paul B., 767  
 Steinberg, Israel, \*369, \*388, \*440,  
 \*653, \*840, \*874, \*901  
 Steiner, R. E., \*279  
 Stephen, David, 767  
 Stevens, Edward M., \*365  
 Stevenson, Clyde A., \*777  
 Stoll, Basil A., 766  
 Storaasli, J. P., 774  
 Straube, Kurt R., \*222  
 Suby, Howard I., 259  
 Suker, J. R., 513  
 Sumerling, M. D., \*451  
 Sun, Donald I.-C., \*461  
 Sundin, T., 518  
 Sutherland, Jerome D., \*416  
 Sutow, W. W., 1031  
  
 Takekawa, Shoichi D., \*800  
 Talledo, E., 516  
 Taplin, G. V., 1027  
 Taybi, Hooshang, \*782  
 Tazumi, Masahiro, \*595  
 Tefft, M., \*675  
 Ten Cate, Harry W., 773  
 Ter-Pogossian, Michel M., 521

- Teschendorf, W., 1020  
 Thaggard, Alvin, Jr., 517  
 Thomas, Sydney F., \*318  
 Thompson, D. W., 512  
 Tiene, E. Schwarz, 513  
 Ting, Y. M., \*343  
 Titus, Jack L., \*800  
 Trackler, Richard T., \*160  
 Trusler, G. A., \*378  
 Tulloch, W. Selby, 261  
 Tye, James G., \*434  
 Ulrich, J., \*239  
 Unger, Sol M., \*616  
 Utz, David C., \*114  
 Vargas S., Jorge, 766  
 Vennart, J., 266  
 Viamonte, M., Jr., \*723  
 Vigne, J., 770  
 Vleugels, E., 769  
 Vogelsang, H., 769  
 Volpe, Joseph A., 1029  
 Voltz, Phillip W., Jr., 517  
 Waddington, Margaret, \*611  
 Wagner, P., 1027  
 Wagoner, Earl V., Jr., \*487  
 Ward, G. M., 1031  
 Webster, J. R., Jr., 513  
 Weens, H. S., 516, \*672  
 Weibel, Jorge, \*660  
 Weijer, D. L., 1030  
 Weiss, Thomas E., 523  
 Weiss, William, \*404  
 Weller, S., 773  
 Wettstein, P., \*436  
 Wheeler, Paul C., \*365  
 Wiener, Stephen N., \*859  
 Wierny, L., 262  
 Wiesner, Jerome J., 517  
 Wigdahl, Luther O., 1026  
 Wigglesworth, F. W., 767  
 Williams, Clyde M., \*225  
 Williams, Ralph H., \*79  
 Wilson, Charles B., \*570  
 Wilson, D. W., 1031  
 Wise, Robert E., 773  
 Wishard, William Niles, \*215  
 Wishard, William Niles, Jr., 1023  
 Wisoff, Carl P., \*63  
 Witten, David M., \*114  
 Wolfel, Donald A., \*334  
 Wolff, Hans, 264  
 Wolff, Lawrence J., \*365  
 Wollschlaeger, Gertraud, \*599  
 Wollschlaeger, Paul B., \*599  
 Wolman, Lionel, 1022  
 Woodcock, Gillian E., 266  
 Workman, Joseph B., 1026  
 Young, Lawrence E., 259  
 Zaunbauer, W., 768  
 Ziedses des Plantes, B. G., 773  
 Zingesser, Lawrence H., \*535  
 Ziter, Fred M., Jr., \*291  
 Zuppinger, A., 1024  
 Zuspan, F. P., 516

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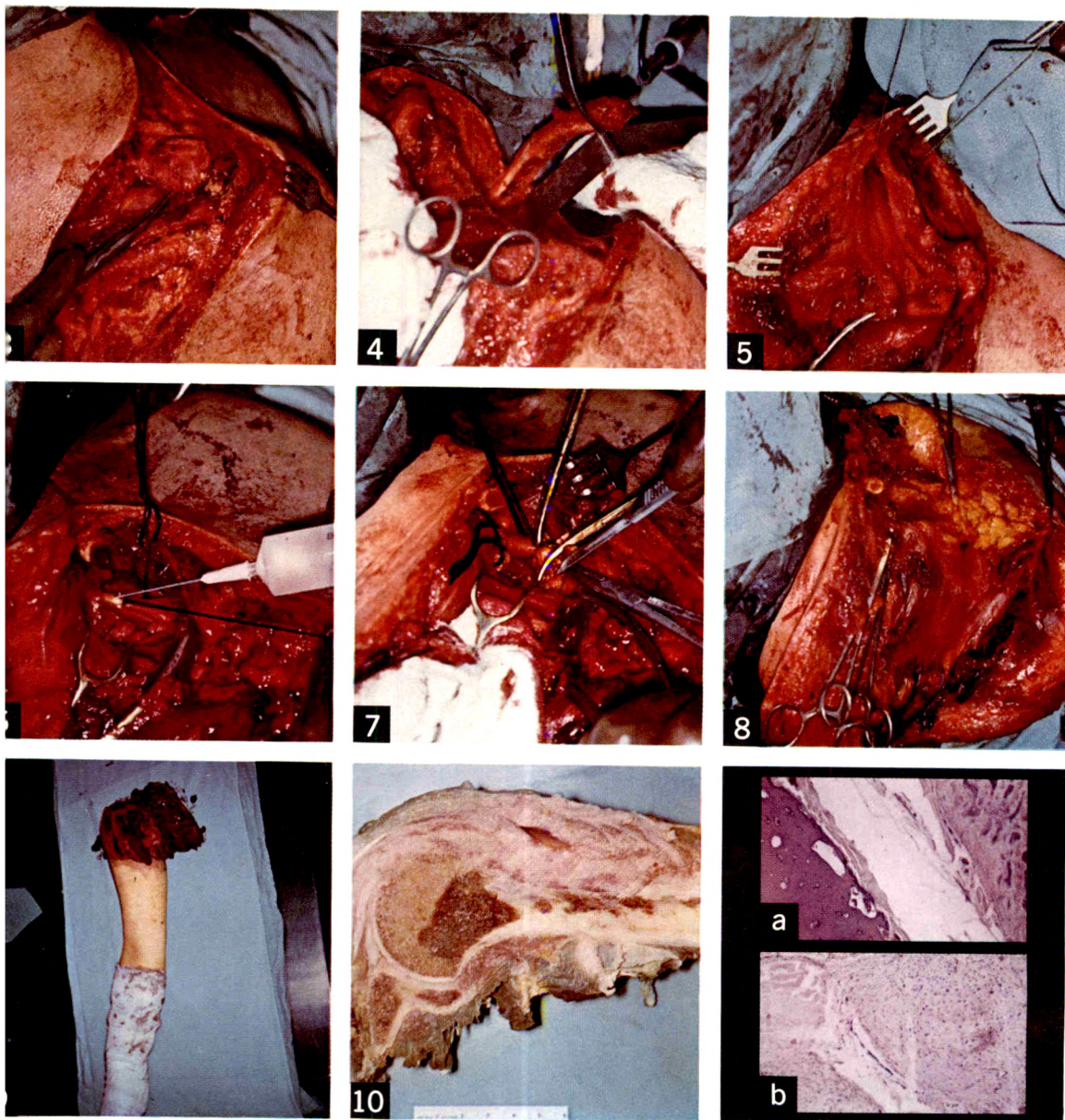
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Figure 1. (Prebiopsy) Radiographic study disclosed extensive cuff-like, irregular calcification surrounding the proximal humerus with involvement and increasing density of the adjacent bones.

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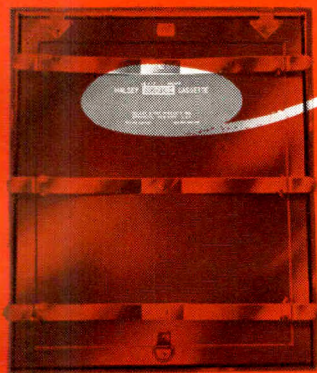


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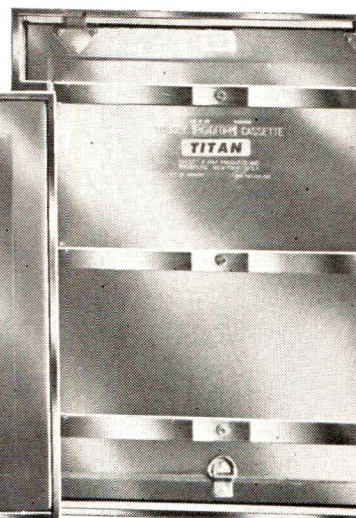
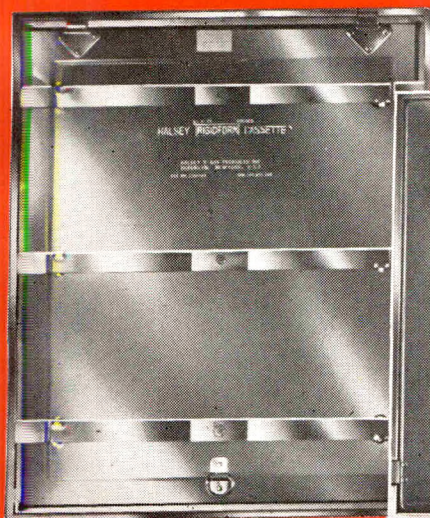
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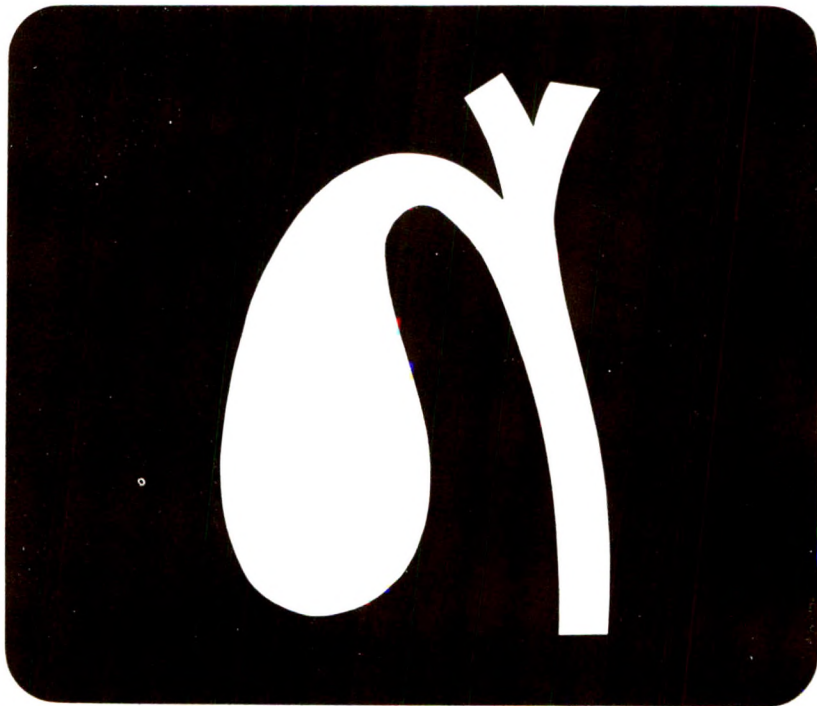
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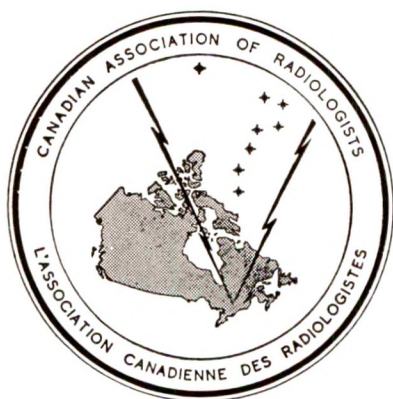
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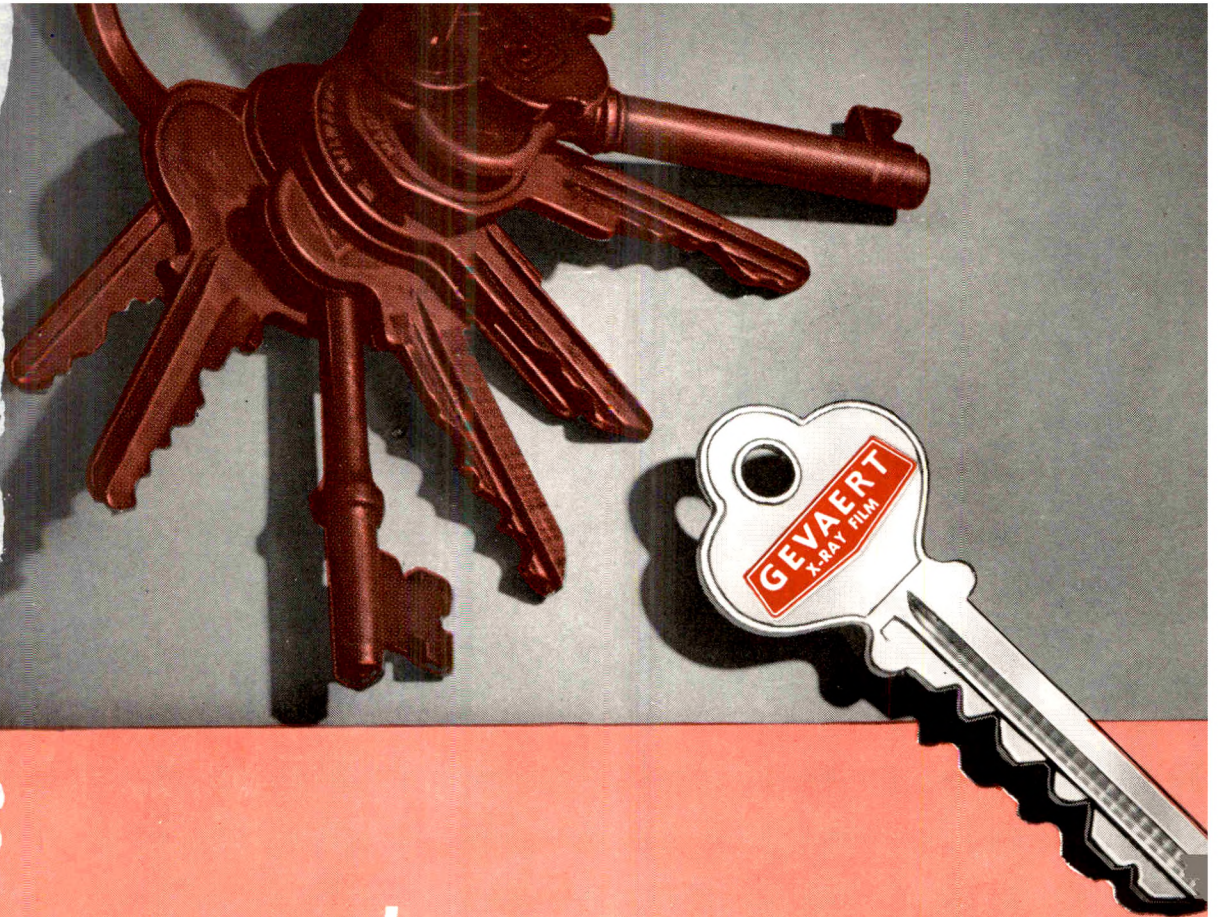
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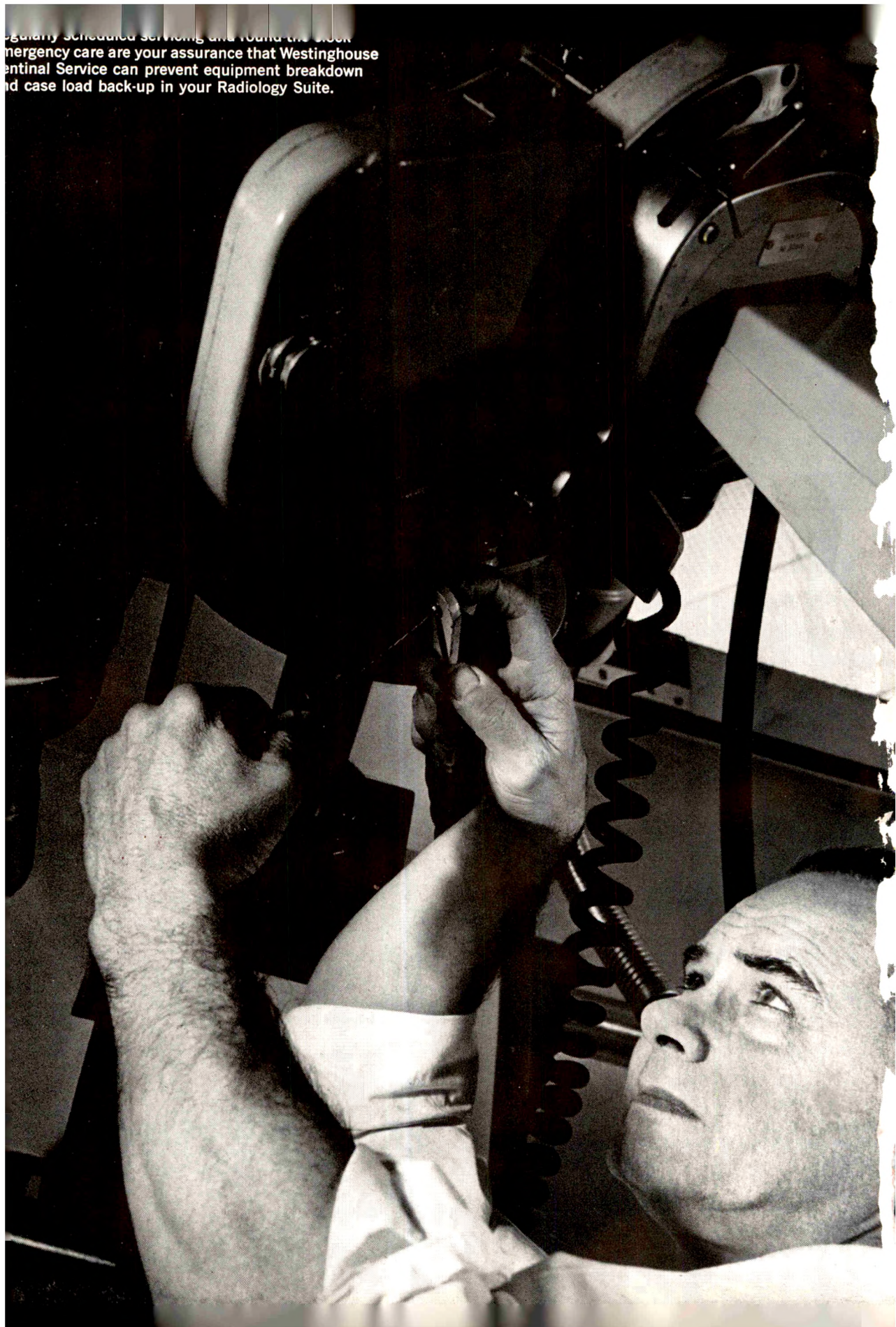
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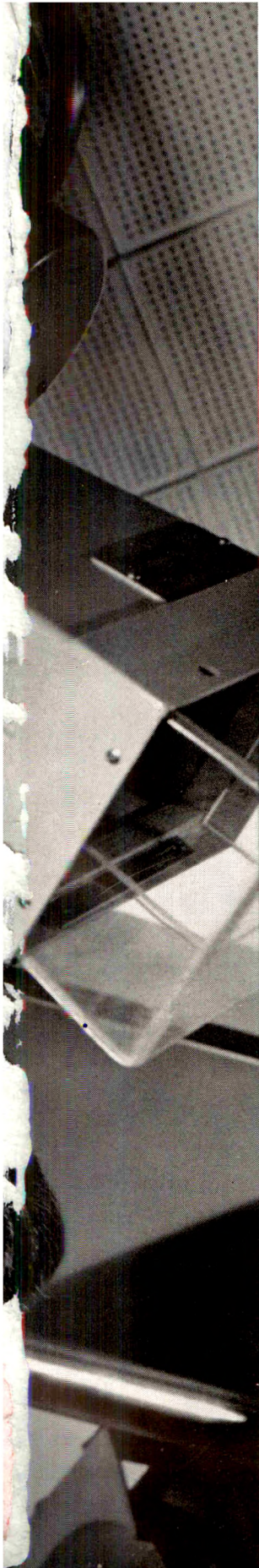
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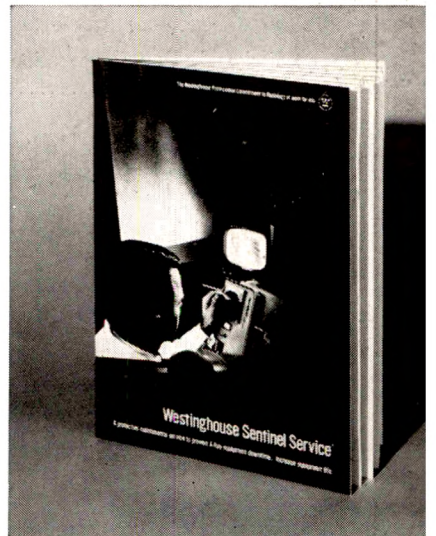
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# INDEX TO ADVERTISERS

Atomic Energy of Canada Ltd. ....	vii
Barnes-Hind Barium Products ....	xliv
Dunlee Corporation ....	xxiii
du Pont, E. I., de Nemours Co. ....	xvi, xvii
Eastman Kodak Company ....	xli, xlii, xliii, xlv
E-Z-Em Company ....	xviii
General Aniline & Film Corporation ....	ix
General Electric Company ....	xix, xx, xxi, liii
Halsey X-Ray Products Company ....	xlv
Hanley Medical Equipment Company ....	xxxviii
High Voltage Engineering ....	Second Cover
Hospital for Joint Diseases & Medical Center (New York City) ....	xxxviii
Keleket X-Ray Corporation ....	xiv, xv
Lafayette Pharmacal, Inc. ....	xiii
Liebel-Flarsheim Company (Div. of Ritter-Pfaudler Corporation) ....	xxvii
Low X-Ray Corporation ....	xlix
Machlett Laboratories, Inc. ....	xxix, xxx
McElroy, Donald, Inc. ....	xlvi
M.E.L. Equipment Company Ltd. ....	vi
North American Philips Company ....	xxiv, xxv
Nuclear-Chicago Corporation ....	viii
Physicians Technical Equipment Company, Inc. ....	xxxviii
Picker X-Ray Corporation ....	xi, xxii, xxvi, xxxi, Fourth Cover
Profexray, Inc. ....	i
Schick X-Ray Company ....	x
Siemens Medical of America, Inc. ....	xxxvi, xxxvii
Squibb, E. R., & Sons, Inc. ....	v, xxviii, xxxiv, xxxv
Standard X-Ray Company ....	xii
Thomas, Charles C., Publisher ....	liv
Veterans Administration Hospital (New York City) ....	xxxviii
Westinghouse Electric Corporation ....	i, li
Winthrop Laboratories ....	xxxii, xxxiii, xlv, xlvii
Wolf X-Ray Corporation ....	Third Cover



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